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Don't Leave a Vacuum

Nature abhors a vacuum, yet too often we have created a vacuum which is most certainly going to be filled by someone else if we of the medical profession do not act.

I am referring here to the disinclination of some of our county medical societies to name a cancer committee, and a subsequent lack of cooperation with the American Cancer Society in establishing a county medical society group as the professional education committee of the local cancer unit.

The consequences of our lack of action, or indifference to the needs of the various health groups, may reach further than most of us apparently realize. Numerous cases can be cited where osteopaths, optometrists, chiropodists, and in one instance a chiropractor, were asked to serve on local American Cancer Society boards after the medical society refused to act, or after accepting committee responsibilities, were indifferent to the lay needs for medical advice.

As Dr. J. Layton Cochran pointed out in his article in the July, 1957, issue of the *Journal*, state and national organizations are predicated upon medical participation. Local laymen, who may not be aware of their state and national reliance upon the medical profession, and who may not be aware of the difference between a number of people they may habitually call "doctor," may not realize the difficulties into which the organization they are sponsoring may fall if the distinction is not clearly made at the inception of the organization.

Our profession has spent considerable sums for public relations experts to tell us how to better our relations with our communities. Cooperation in serving the various health organizations to our best ability will provide a stepping-

stone that few of us can afford to ignore. The need for medical advice in lay health organizations is ever present. If the need is to be filled by competent members of the profession, it is up to us, through county medical societies, to see that the opportunity is not lost.

—JOHN F. THOMAS, M.D., Austin.

Observance and Challenge

". . . Therefore, I, as Governor of Texas, do hereby designate the week of April 20-26, 1958, as Medical Education Week in Texas, and urge all of our citizens to recognize the common debt to the medical schools and to foster their continued freedom and security."

—Price Daniel.

Joining with states across the Union, Texas is celebrating the week of April 20 as Medical Education Week.

Fittingly enough, members of the Texas Medical Association begin the week discussing in their House of Delegates more than one subject relating to medical education—the best location for a proposed new medical branch of the University of Texas and whether or not Association members should be assessed to assure financial support of the American Medical Education Foundation, for example.

County medical societies were encouraged to give such recognition to Medical Education Week as seems appropriate in their communities, and several announced their plans to call the observance to the attention of fellow townsmen.

The United States today has 82 approved medical schools, 78 of them granting degrees of doctor of medicine and 4 having 2 year basic medical science courses. In addition, there are 4 newly developing schools of medicine, and 1 basic science school is undergoing transition to a full 4 year program. In the year 1930-1931, there were 76 schools with 21,982 students and 4,735 graduates; in 1956-1957 there were 82 schools with 29,130 students and 6,796 graduates. Individual applicants to medical schools, which had decreased from 24,434 in 1949-1950 to a low of 14,538 in 1954-1955, had climbed to 15,918 in 1956-1957. In addition to teaching students seeking M.D. degrees, almost every institution conducts courses for dental, pharmacy, nursing, intern, residency, postgraduate, and other students. In 1956-1957 undergraduate medical students totaled 29,130, as noted previously, whereas other students numbered 62,964.* Simultaneously, medical schools are carrying on research of major importance.

This intricate system of education that is responsible for turning out the doctors of tomorrow is faced with a mass of problems and challenges. Changing dimensions of medical knowledge have an impact on such aspects as the time required for a medical education and the cost of such education to student and institution. Curriculum also needs reevaluation and perhaps revision. Staffing is made difficult because of competition from industry, private practice, and inadequate financial support—331 unfilled positions in budgeted full-time faculty positions were reported by approved medical schools in 1957.* Ability of present facilities to meet the needs for professional personnel in the future is in question not only in Texas.

Medical Education Week can serve to focus the attention of everyone, especially the general public, on the service medical schools have provided and are providing in the interest of high class medical care and furtherance of knowledge of health and disease. It also can direct attention, particularly of the physician who can recognize readily the ramifications of such problems, to the challenges which face medical institutions and cry for forceful leadership and creative understanding.

**Resumé of Status of Medical Education in the United States, Prepared for Annual Meeting of State Chairman—American Medical Education Foundation, January 25-26, 1958.*

Sugar Coated Pill?

Beginning with this issue, the *Journal* will have a monthly feature giving information about new drugs coming on the market. The material is prepared by Herbert Schwartz, special instructor in pharmacy at the University of Texas in Austin. The comments on drugs can be found in the News Section.

Mr. Schwartz, who is a graduate of the University of Pittsburgh with a master of science degree from the University of Texas, has offered to provide this information on drugs of current interest to the medical profession if readers of the *Journal* want it. He also is author of a column in the *Texas Druggist,* periodical of the Texas Pharmaceutical Association.

It is planned to use this feature for several months on a trial basis. If readers like it, it will be continued; if not, it will be discontinued. Comments about the types of drugs included, the facts given about them, and whether or not such a contribution is of value will be appreciated.

Bigger and Better

Initiative in providing excellent programs in addition to those routinely held as a part of the Texas Medical Association annual session came from two Association committees not usually concerned with the session. The Committee on Nuclear and Atomic Medicine chose the 1958 annual session as a jumping off place for its project of education of Texas doctors in the problems of peacetime and wartime uses of atomic energy by having a symposium in which experts working with the Atomic Energy Commission were given a prominent place. The Committee on Cancer planned a conference emphasizing the facilities and services available in Texas for care of cancer patients. Both of these meetings were arranged with practicing physicians in mind.

Requesting permission to take advantage of the presence of physicians at the annual session, the University of Texas Postgraduate School of Medicine, Baylor University College of Medicine, the Texas State Department of Health, and the United States Public Health Service set a National Venereal Disease Postgraduate Conference with speakers from throughout the country in Houston immediately following the session.

These various symposiums and conferences, together with the many related specialty society activities now considered a major attraction, the orientation program for new members held this year for the first time in conjunction with the session, and the various traditional facets of the program proper have meant a tremendous concentration of talent and subject matter for the benefit of attending physicians. With almost the entire array of events offered without fee, the physician really has a bargain.

It is truly encouraging to note the enthusiasm of many groups and individuals, the pooling of speakers and financial resources, the cooperation in scheduling which can result only in an exceptionally fine session and a spirit of unity.

★ Current Editorial Comment

Cancer in Children

Cancer among children is at once the most discouraging and the most helpful segment of the total cancer problem. It is discouraging because it is the major disease causing death in the 5 to 14 year age groups and second only to pneumonia in the group under 4 years. It is encouraging because it is in the types of cancer occurring among children that the first break in the cancer riddle may occur. If this does

This department of the JOURNAL *presents editorial comments on current items pertaining to the science, art, and practice of medicine, contributed by members of the Texas Medical Association and scientists closely associated with the medical profession of Texas. Invitation is hereby extended to any member of the Texas Medical Association to submit such discussions for this department. The discussions should not be more than 500 words in length.*

happen, the restoration to normal health may be expected to be as dramatic as it was among diabetic children less than 50 years ago, for children have not developed the degenerative processes incidental to maturity, and the control of the disease cancer, as of the disease diabetes, removes the sole disability.

Children have been treated at the University of Texas M. D. Anderson Hospital and Tumor Institute since the institution opened. The type of care and the facilities needed to keep the children comfortable and happy are so specialized that a separate pediatric ward was activated. Approximately 100 new patients with malignant neoplastic disease were seen last year, representing a steady increase over preceding years.

In the period between July 15, 1954, and January 1, 1958, there were 243 children with malignant neoplastic disease referred to the Pediatric Service of the University of Texas M. D. Anderson Hospital and Tumor Institute. During the same period, 45 patients with hemangioma or lymphangioma and 139 other children with benign neoplasms or nonmalignant conditions were seen.

Table 1 summarizes the types of malignant and neoplastic diseases found among children at the University of Texas M. D. Anderson Hospital and Tumor Institute.

Acute leukemia and the lymphomas are being studied intensively, not only from the standpoint of improvement in chemotherapy and trial of new and active drugs but in the management of the complications of the disease. This emphasis undoubtedly accounts for many of the referrals. This series is made up largely of Texas children.

Great strides are being made in prolonging with comfort the lives of these children by the application of the findings of the rapidly unfolding field of chemotherapy. These findings are made available to referring physicians as rapidly as they are known. The family physician plays a genuine role in the attack on childhood cancer, as he first of all is the person responsible for the detection of the malady. The problem of finding curative measures for cancer and leukemia is one that involves the cooperative effort of parent and child, family physician, and the clinical and research centers where dedicated men and women are studying these diseases.

TABLE 1.—*Types of Malignant and Neoplastic Disease Among Children at the University of Texas M. D. Anderson Hospital and Tumor Institute.*

Type of Disease	No. Cases
Acute leukemia	69
Chronic leukemia	2
Lymphosarcoma	25
Hodgkin's disease	16
Neuroblastoma	15
Retinoendothelioma	7
Wilms' tumor	10
Retinoblastoma	8
Brain and central nervous system sarcoma	10
Soft tissue sarcoma	23
Ewing's sarcoma	14
Osteogenic sarcoma	12
Other bone sarcomas	3
Miscellaneous malignancies	29
Hemangioma or lymphangioma	45
Benign tumors	61
Non-neoplastic disease	78
Total	427

—GRANT TAYLOR, M.D., Houston.

Dr. Taylor, M. D. Anderson Hospital, Texas Medical Center, Houston 25.

It's April Again —

Time to Attend

Texas Medical Association Annual Session

April 19-22 **Houston**

— Program in March Journal

President's Page

My Last Message

The theme of the Texas Medical Association for this administrative year has been "For a Better Informed Membership." Your home office staff, your legal counsel, and your elected officials have crossed Texas many times this year carrying messages and information to the membership of this state. Hundreds of informative books and pamphlets have been mailed to the official representatives. Many pages have been written, and with considerable effort, dealing with different subjects which should concern the busy physicians of Texas.

As the year ends and our accomplishments are tabulated, we feel a brief but humble sense of gratitude. This is short lived, however, for as we enumerate the many things we have been able to do, a feeling of futility or frustration hovers over us like an autumn cloud.

Any fair minded person who spends a year mingling with and talking to the physicians throughout this state will be convinced that the medical needs are being met by as well trained and dedicated a group of physicians as can be found anywhere else in the world. Most of these men are playing increasingly important roles as citizens in their respective communities. In the most remote areas, physicians are striving to improve the standards of medicine and surgery and are doing a masterful job of keeping abreast of the discoveries which are coming so rapidly. Every effort is being made to correct or punish the small number who through carelessness or dishonesty would debase the good name of this noble profession.

In normal times this would be enough to ask of any group of people. Unfortunately these times are not normal. Spreading over the world is an atheistic ideology which is sworn to the task of destroying Christianity and all other forms of religion. The average American today in his seemingly secure position can see no reason for concern. He sincerely believes that any threat of communism is so far away that it can never affect him. Therein lies the strength and success of a group of Marxists which numbered a mere two dozen in 1903. In 1917, they had only 40,000 followers. Today they have absolute control over more than 900,000,000 people. This is one-third of the population of the earth. The United States is their coveted goal. A

brief insight into the inroads they have made into almost every phase of American life, including our own profession, will startle the most complacent.

Can it be that the average American today is not as interested in preserving his freedom and passing it along to his children as were the patriots at San Jacinto, the Alamo, and Valley Forge? Have success and the many comforts of life softened and blinded him to the necessity of jealously guarding our freedom?

Too many people have shown concern and have requested sources of reading material for me to believe that we have passed the point of no return. For their benefit, several sources from which anticommunist and conservative literature can be obtained are listed below. Since the hour is late and our freedom is so badly jeopardized, may every doctor in Texas avail himself of some of this literature and carry it in his pocket until he has finished each publication. Only a well informed citizenship can cope with these godless, dedicated fanatics.

Denton Kerr

Sources of Conservative Information

1. Christian Anticommunist Crusade, Box 890, Long Beach, Calif. (Send $5 or $10—enough to be placed on mailing list.)
2. Human Events, 835 Case Street, N.W., Washington 6, D. C. ($10 per year.)
3. Association of American Physicians and Surgeons, 185 North Wabash Avenue, Chicago 1. ($20 for an annual membership and an informative weekly newsletter.)
4. Challenge to Socialism, 9127 Jones Mill Road, Chevy Chase 15, Md. ($4 per year.)
5. National Education Program, Searcy, Ark. (Ask to be placed on mailing list and send small donation if you wish.)
6. Dan Smoot Report, P. O. Box 1305, 1430 San Rafael Drive, Dallas. ($10 per year.)
7. Economic Council Letter, Empire State Building, New York 1. ($10 per year.)

ORIGINAL ARTICLES

Tumor Seminar

Conducted by

MALCOLM B. DOCKERTY, M.D.

Rochester, Minnesota

THE thirteenth annual tumor seminar of the San Antonio Society of Pathologists at Brooke Army Hospital, Fort Sam Houston, Texas, on November 3, 1956, was opened by Dr. Louis J. Manhoff, Jr., president of the society, who acknowledged the generous support given by the South Central Regional Committee, College of American Pathologists and the Texas Division of the American Cancer Society.

Dr. Malcolm B. Dockerty, member of the Mayo Clinic staff, conducted this seminar for the San Antonio Society of Pathologists at Fort Sam Houston on November 3, 1956.

The commanding general of Brooke Army Medical Center, Major General William E. Shambora, MC, and the commandant of the Army Medical Service School and moderator of the seminar, Brigadier General Elbert DeCoursey, MC, were introduced in turn. General DeCoursey introduced Dr. Malcolm B. Dockerty, Mayo Foundation, Rochester, Minn., conductor of the seminar.

CASE 1

Diagnosis.—Carcinoid of duodenum.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 55 year old woman 6 years past menopause had had uterine bleeding of 2 months' duration. She also gave a history of belching, indigestion, nausea, vomiting, and an occasional dull ache in the epigastrium of several years' duration. Dilatation and curettage revealed adenocarcinoma of endometrium, for which the patient received radium and 6 weeks later had a panhysterectomy. At the time of surgery an apparently superficial hard tumor, 2 cm. in diameter, was found in the wall of the duodenum near the pylorus. After removal of the tumor and report of biopsy a subtotal gastrectomy was performed, together with further removal of portions of the duodenum.

Dr. Dockerty: There are two roentgenograms on the first case, and Col. Henry C. Harrell, MC, radiologist at Brooke Army Hospital, has kindly consented to discuss them. I might add that in this case, as with the other cases having roentgenograms, Colonel Harrell has had no opportunity to study them, and he is further handicapped in having only a reproduction to interpret.

Colonel Harrell: You notice there is a sort of a "3" shaped sign which gives the impression that there is a little tumefaction extending knucklelike into the duodenum. The cap is large, so it suggests obstruction and tumefaction in the wall. As far as a differential diagnosis goes, from the x-ray one would consider an enlargement of Brunner's glands, which can do this, but that is usually all around; carcinoid of course is a possibility, as well as some other tumor of the small bowel wall. The mucosa cannot be made out over the tumefaction too well. I think perhaps the mucosa may be destroyed.

Dr. Dockerty: I am glad the colonel went over these roentgenograms, because the older textbooks told us that carcinoid tumor—I am giving you an inkling now as to what my diagnosis is—that carcinoid tumor was an asymptomatic plaything of the pathologists which for the most part was confined to the tip of the appendix. Many of these extra-appendiceal cases, however, are symptomatic.

Under low power we see that the neoplasm is submucosal and that the mucosa is intact over it. These growths are supposed to come from the Kultschitzky cells at the base of the crypts of Lieberkühn, and it is remarkable that perhaps only 1 case in 10 of a carcinoid tumor will exhibit mucosal ulceration to the extent that the patient will bleed, as patients will bleed from gastric and duodenal ulcers, myomas, and so on. Clinically, they do obstruct, however.

I think we can rule out immediately the possibility that this growth is a metastasis from the patient's endometrial carcinoma. The latter was not particularly invasive in the sections I examined. This also would be a most unusual site for a solitary metastasis. Such growths, when they spread, will go to nodes or spread by peritoneal sedimentation. The scirrhous reaction in this growth would be unusual for an endometrial carcinoma, and the composition of prismatic cells containing pink cytoplasmic granules is

unusual for metastatic tumor from the uterus. The duodenal tumor is a carcinoid to anyone who has seen more than one or two examples, and we do not need silver and "gold" stains to prove our point.

I would like to say a word about carcinoids in general, and duodenal carcinoids in particular. As far as I am concerned, carcinoid tumors are *all* cancers, regardless of location or size. Given time, in my opinion, every carcinoid tumor eventually will metastasize. Others in the literature have also come to this conclusion, which is at variance, of course, with the notion taught in the older textbooks, that carcinoids were carcinoma-like, microscopically, but that they did not behave clinically like cancers at all. In our experience 13 of our last 16 carcinoids of the small intestine were associated with metastasis, and in the literature the figure of incidence for the spread of extra-appendiceal carcinoids is about 35 per cent. The ileal ones have a greater tendency to metastasize than do the rectal or the gastric or the duodenal ones, and as I say, the figure there will run perhaps more than 50 per cent in general. Now the appendiceal growths are small. If you go over the age incidence for people with appendiceal carcinoid tumors, you will find that the patients are young (between 20 and 30). It appears that these little growths, which are sometimes located proximally in the appendix, but more frequently at the tip, perhaps will produce mild symptoms of obstruction, and they are removed, because of those symptoms, early. In the ileum, however, where they have more of a chance to grow before they produce any symptoms, they metastasize, and the average age for patients with ileal carcinoids is around 50 to 60, or perhaps I should say 40 to 60. So the factor of size is very important, and the age factor closely parallels it. In the good old days proctologists were prone to fulgurate the submucosal nodules in the rectum without the benefit of a biopsy, but nobody ever got into trouble with them. Nowadays proctologists are excising them for microscopic examination, and we are finding in our clinic that rectal carcinoids actually are more frequent than appendiceal carcinoids.

It is important to remember that the lump that you feel in the patient with a carcinoid tumor is the metastasis and not the primary. This is characteristic. I will not accept as carcinoid any primary lesion in the bowel that is over 1 inch in diameter. As regards the metastatic nodules, however, I have seen 2,000 Gm. of liver metastases from a 1 Gm. ileal carcinoid, which in metastasizing also involved the root of the mesentery, completely blocked off the mesenteric vessels, and killed the patient by infarcting along one segment of bowel.

When you obtain such a large bulk of carcinoid tumor from a metastatic lesion, you are likely to have the associated carcinoid syndrome recently described by Biörck, Thorson, and others. This syndrome consists of an intractable diarrhea which at times suggests that the patient has a gastrocolic fistula. Patients have palpitation, flushing, and cyanosis, with patchy reddish discolored areas over their bodies. Eventually signs of right heart failure ensue from subendocardial fibrosis that may kill the patient. There are peculiar signs on electrocardiographic studies. Pulmonary hypertension and asthma are also common in the syndrome. If you analyze the urine for serotonin, which is the product of the normal "carcinoid" cell (or Kultschitzky cell), you will find that its end product, 5-hydroxyindoleacetic acid, is present in large amounts. Accordingly, it now becomes possible to diagnose carcinoids preoperatively.

Since it is possible, therefore, for a carcinoid tumor with metastasis to kill the patient by ruining the right side of his heart, it becomes the duty of the pathologist to make the diagnosis by fresh frozen sections where possible, so that the surgeon may deal with it and its metastasis appropriately. In other words, it is recommended that a partial hepatectomy be done for a large metastatic carcinoid tumor in the liver, or that other similar metastatic masses of carcinoid tumor be excised as completely as possible.

The location of carcinoids in the duodenum, as in this particular nodule, is rare. As far as I could gather, there are only 9 duodenal carcinoids in the literature. This compares with an incidence of about 25 carcinoids of the stomach, hundreds from the ileum, and perhaps 50 from the rectum. Seven of the 9 duodenal examples have metastasized. Only 1 duodenal carcinoid in the literature has produced symptoms, and this case is probably another such example.

Submitted Diagnoses.—Carcinoid, 41; adenocarcinoma, 2; islet cell tumor, 1; islet cell carcinoma (in ectopic pancreas?), 1.

Dr. Dockerty: We notice that most persons submitting diagnoses thought this was a carcinoid. As for adenocarcinoma, in the literature I have called these "adenocarcinoma, carcinoid type," to emphasize that carcinoids were cancers, but have called them grade I to indicate that they were low grade, well differentiated, and slowly growing cancers. Islet cell tumor is an interesting diagnosis. Islet cell tumors microscopically look much like carcinoids, but I think that pathologists sometimes have to be philosophers, and they have to be biased a little bit by the clinical history. If this patient had a blood sugar of 10 mg., I probably would have called this an islet cell tumor. The two are very similar, and in the literature islet cell tumors frequently have been confused with carcinoid tumors. I might say that 1 per cent of islet cell tumors occur in ectopic locations.

Dr. Leo Lowbeer, Tulsa, Okla: We had a 32 year old male patient who had a microcytic hypochromic

anemia and occult blood in the stool. The roentgenograms of the gastrointestinal tract were entirely negative. The surgeon did an exploratory laparotomy, and at first found nothing; but since there was so obviously loss of blood from the intestinal tract, he went carefully through all small intestinal loops until he got a tiny nodule, which he excised, and which proved to be a small (less than 5 mm.) carcinoid which was ulcerated, and the only symptom of which was bleeding.

Dr. Dockerty: Our experience has been the same. I mentioned the figure of 10 per cent. We had 1 patient who presented with the problem of severe exsanguinating gastrointestinal hemorrhage, with negative x-rays. His carcinoid was as large as a thumbnail and was ulcerated. It seems that these carcinoids have a scirrhous reaction, which was shown in this case, and they are not very vascular. You will find microscopic ulceration much more frequently than you will discover gross excavation.

Dr. A. O. Severance, San Antonio: In February this patient was last seen by the family doctor, and at that time there was no clinical evidence of recurrence or metastasis. The Fourth Army Area Medical Laboratory analyzed his urine and failed to get any indication of an increased serotonin level.

Dr. Dockerty: Well, that's comforting, because these growths may have occult metastases which take a long time before they become palpable, and while they are doing this the patient's physical condition can deteriorate from right heart failure. Postoperative urinalyses for 5-hydroxyindoleacetic acid are indicated to rule out the possibility of removable metastases which are otherwise undetectable while they are still within reasonable size.

Dr. J. H. Childers, Galveston: How often do you see cells in this particular type of tumor which produce mucin?

Dr. Dockerty: I have seen and reported such in a case of carcinoid of the terminal ileum. About 2 per cent of them have real stainable mucus. Incidentally, I am glad you brought that point up. One reason for my labeling these tumors "adenocarcinoma, carcinoid type," was that in addition to forming little pseudorosettes, I felt that they could also actually be mucus-producing tumors; but you won't find it in many of them.

CASES 2 AND 3

Dr. Dockerty: I would like to discuss cases 2 and 3 together, since they are both papillary breast lesions.

CASE 2

Diagnosis.—Benign papillomatosis of the breast.

Contributors.—Dr. D. A. Todd and Dr. A. M. Richmond, Nix Hospital Clinical Laboratory, San Antonio.

History.—A 48 year old white woman for 7 months had noted a bloody discharge which could be expressed from the right nipple by pressure over a small nodular mass 1 cm. to the right of the areola. There was no discharge from the left nipple. A mass had been removed from the left breast many years before. Smears of the secretion showed only red blood cells.

CASE 3

Diagnosis.—Papillary cancer of the breast.

Contributor.—Dr. N. H. Jacob, Jr., Santa Rosa Hospital, San Antonio.

History.—A 64 year old white woman had a simple mastectomy for a lump in the right breast which had been growing slowly for the past year, reaching the size of a fist.

Dr. Dockerty: In the first place, case 2 is obviously a papillary lesion of the breast consisting of anastomosing, epithelium-covered papillary fronds which come together to produce numerous glandlike spaces. The growth seems to be multicentric and involves several large dilated ducts. The connective tissue cores of these villous projections actually make up the bulk of the tumor. Dark staining, prismatic cells that have the microscopic appearance of ordinary breast duct cells are lined up with little stratification. Mitotic figures are few and far between. The growth is reminiscent of an intracanalicular fibroadenoma, with the important difference that most of the proliferation is on the part of the epithelium rather than of the connective tissue. In some areas the cells are considerably larger, they are granular, and the granules are a little bit eosinophilic; these are of apocrine origin.

By contrast to the picture in case 2, case 3 shows a growth which, although papillary, is more solidly epithelial, with the connective tissue stalks of the villi being much more tenuous. In some areas connective tissue is in fact absent, and the epithelium displays a cribriform pattern which, in places, is reminiscent of the picture seen in comedocarcinoma of the breast. Here ducts become dilated and papillations converge on the center of the duct like the spokes of a wheel, and one frequently finds necrosis. As a matter of fact, in comedocarcinoma one frequently finds a papillary epithelial component. As contrasted with case 2, the epithelial component in case 3 is cytologically active and anaplastic, with an average increase in cell size and marked nuclear hyperchromatism. Nucleoli are prominent and mitotic figures are easy to find. There is invasion of the "capsule," and admixture with apocrine cells is lacking.

By way of discussing these two interesting and somewhat similar cases, we can divide papillary growths of the breast in general into three categories: papilloma; papillary cancer; and a third, recently described by Jones in an article titled "Florid Papillomatosis of the Nipple Ducts." The malignancy line differentiating the good from the bad in these papillary tumors is an undulating one, and almost every pathologist has his mind made up pretty definitely about what he is going to call benign and malignant.

I always maintain that "A man convinced against his will is of the same opinion still," and I'm not going to try too much to change your minds. However, I will give you my philosophy on these things. In general I should say that papillomas of the breast are overtreated, and I am sure that papillary cancers of the breast are undertreated. In the past at our clinic I have been impressed by the fact that about 50 per cent of papillomas, the so-called benign ones, are multicentric. Perhaps half of these will occur right under the nipple; sometimes they are palpable, sometimes not; sometimes there is a bloody discharge, and sometimes there isn't. I have not been impressed by the thesis that the remaining multicentric papillary growths in this 50 per cent are in fact confined to one segment of the breast. Accordingly, I was not too unhappy over the tendency of our senior surgeons to perform simple mastectomies for papillomas. Some of the younger generation are currently injecting radiopaque dye. They are slitting nipple ducts, and they are cutting "pie" segments out of the breast, while they maintain that they, too, are getting 100 per cent cures. Thus the treatment has swung, certainly at our institution, toward a much more conservative approach to papilloma of the breast.

On the other hand I have always advocated radical mastectomy for what I call honest-to-goodness papillary cancer. In 3 instances I have seen nodes involved, and in 3 additional cases I have seen the patients die as a result of extensive local recurrence and chest wall invasion following simple mastectomy. One such recurrence took 6 years to make itself manifest.

What are some of the gross differences between papilloma and papillary cancer? In papillomas there usually is no lump, but if there is one it is a tiny nubbin right under the nipple. About 50 per cent of these are located in the ampulla. There may be a bloody discharge. Papillary cancer frequently forms a lump or occurs in the wall of a cyst. In a recently studied series of 48 cystic cancers of the breast at our institution no less than 33 were papillary in nature.

Microscopically the presence of rather fibrous, thick, heavy stalks with layering of perhaps two cells at most over these stalks; good vascularity of the stalks; the presence of apocrine glands; lack of invasion; and multicentric location under the nipple, all favor the diagnosis of a benign papillary tumor of the breast. In this cancer I think the most important evidence for malignancy is cytologic. If I find pathologic mitotic figures in one of these proliferative growths, it is cancer, in my estimation. In addition, the cells will show the usual signs of anaplasia: hyperchromatism, many mitotic figures, and frequently an over-all comedo appearance.

My diagnosis in case 2 is papilloma, or papillomatosis, and in case 3, grade II papillary adenocarcinoma of the breast.

Follow-Up, Case 2.—Dr. A. M. Richmond, Nix Hospital: The patient is living and well, after 6 years.

Follow-Up, Case 3.—Dr. N. H. Jacob, Santa Rosa Hospital: This patient was operated upon in 1948 and during this past year (1956) there was a recurrence of a nodule in the axilla which showed the same histologic picture as the original lesion.

Dr. Dockerty: I am certainly interested to hear about this result in case 3. That is an 8 year delayed recurrence; without a doubt, though, the cancer was there in 1948. It simply emphasizes that papillary cancers of the breast are slowly growing, but can metastasize just like any other breast cancer.

Submitted Diagnoses, Case 2.—Intraductal papilloma, 23; intraductal papilloma with cystic disease, 12; adenoma, 1; sclerosing adenosis, 1; intraductal carcinoma, 1; intraductal papillary adenocarcinoma, 6.

Dr. Dockerty: In the analyses of case 2 we have essentially 35 for papilloma, benign, and I won't quarrel too much with the term adenoma. As to sclerosing adenosis, you do sometimes find papillary projections in this lesion, but for the most part in sclerosing adenosis you have solid infiltrating cancer-like areas in the breast with a lot of fibrous tissue, which is sometimes hyalinized around these well differentiated ducts. It can be mixed up with papilloma, but I don't think that that is the diagnosis here. Seven persons voted for cancer, but I would not go that far. As I said in the beginning, the borderline of diagnosis between benignancy and malignancy in papillary growths is an undulating one, and some pathologists, to be safe, call them all cancers.

Submitted Diagnoses, Case 3.—Papillary adenocarcinoma, 32; carcinoma, 1; infiltrating adenocarcinoma, 1; ovarian serous cyst–adenocarcinoma, 1; cystosarcoma phyllodes, 1; angioma, Kaposi type, 1; papilloma, 15; intraductal papillary adenoma, 1; adenoma, sweat gland type, 1.

Dr. Dockerty: In case 3 we have 4 voting for cancer and 1 for metastatic ovarian cancer. Of course if you found a growth like this in the ovary, you perhaps would not suspect that it was primary in the breast; you would call it an ovarian cystadenocarcinoma, papillary type, and you would look for psammoma bodies. I have seen a grade I papillary cancer of the ovary metastasize to the axilla, but I have never seen such a cancer metastasize to the breast. As a matter of fact, the only metastatic cancers to the breast I have in my series are a case of Hodgkin's disease and a case of cancer of the prostate. In any large series you will find that the breast is frequently the seat of primary cancer, but very, very infrequently the seat of metastases, and I doubt that an ovarian carcinoma of such a low grade of malignancy could metastasize to the breast.

Cystosarcoma phyllodes, or phylloides, to my mind is a malignant fibroadenoma, and there is a tremen-

dous increase in the connective tissue, rather than the epithelium. Case 3 showed an epithelial proliferation, whereas cystosarcoma phyllodes is a connective tissue proliferation. One per cent of fibroadenomas are malignant, and perhaps 5 or 6 per cent of the giant fibroadenomas are malignant. I think that we should do away with the sarcoma part of cystosarcoma phyllodes. Clinically, of course, they act like sarcomas; they ulcerate the skin, and the pathologist has great difficulty convincing the clinician that the growth is not a "bad cancer," but a surprisingly small percentage of them actually are cytologically malignant. When they metastasize it is almost 100 per cent by the blood stream, and they metastasize usually as straight sarcomas. I have seen only 2 cases of carcinoma in fibroadenomas.

The lesion in case 3 was vascular, but I think that its epithelial nature is fairly definite, so that I would not consider angioma, Kaposi type. The rest of the diagnoses were those of benign lesions, but I believe that this lesion is definitely a cancer.

Dr. J. J. Andujar, Fort Worth: I note that you graded this as grade II. Are there two grades, grade I, in which you cannot be sure it is malignant, and grade II, all the others, or do you call them grades I, II, III, and IV?

Dr. Dockerty: I do not call all papillomas grade I cancer, although I used to a number of years ago. I also recognize grades I, II, III, and IV in the papillary lesions.

Dr. Andujar: You disagree, then, with the original work of Dr. Broders in which he called them grade I carcinomas?

Dr. Dockerty: I do, to some extent. I have seen grade I papillary cancer of the breast metastasize. I have never seen a growth like the lesion in case 2 metastasize.

General DeCoursey: Many years ago, at the first one of these seminars I ever attended, the philosophy was expressed that when you looked at a breast tumor and weren't sure whether it was benign or malignant, it should be called benign. Have pathologists changed since, or has that philosophy changed since?

Dr. Dockerty: Well, let's put it this way: If you make the mistake of calling benign a borderline lesion—a grade I papillary cancer of the breast—and the surgeon has done an adequate excision around it, the chances are perhaps 95 to 5 that there will be no further trouble.

Dr. T. R. Sunbury, Temple: I called case 2 a grade I papillary adenocarcinoma. About the time these slides came in we had 1 case in which a radical mastectomy was done for a nonpapillary adenocarcinoma, and we found a metastasis in one lymph node which was papillary and did not look any more malignant than case 2. On going back through the breast we found a small papillary adenocarcinoma, which I believe most people here would have called an intraductal papilloma.

Dr. Dockerty: You'll get surprises every once in a while; but you found that papillary cancer after you had found the papillary formation in the axilla, and it was not in the lesion that you first saw in the breast. That brings up the question of how often papilloma is associated with something more serious elsewhere. I am sure that there is a somewhat increased incidence of cancer in breasts that are the seat of papillomas, but I frankly do not believe that if a papilloma is left long enough it is going to become cancer. I will admit that you see the two together once in a while.

CASE 4

Diagnosis.—Benign giant cell tumor of scapula.

Contributor.—Dr. S. W. Bohls, Austin State Hospital, Austin.

History.—A 26 year old white man complained of a lump in the left scapular region and pain in his left arm 6 weeks after receiving an injury. There was a firm, nontender, lemon sized mass on the medial border of the left scapula, half way between the spine and angle. Roentgen ray revealed an irregular, bony, erosive process with the appearance of a malignant lesion at this site. No other abnormalities were noted in the shoulder, and skeletal survey was negative.

Dr. Dockerty: I believe there are two roentgenograms.

Colonel Harrell: This lesion involving the scapula is well described in your protocol. It has too smooth an edge to be a malignant lesion. There usually is more infiltration and irregular destruction of bone in a malignancy, though the part down at the tip does show some irregularity.

General DeCoursey: Is there an x-ray diagnosis?

Colonel Harrell: I'll accept the opinion in the protocol, that this is a malignant lesion, although some of the features of it are not typical.

Dr. Dockerty: There are also certain microscopic features that are not too typical. One is struck, at first, with the tremendous numbers of giant cells; they contain from 10 to 80 nuclei, and they are pretty obviously osteoclasts. They probably arise from fusion of the intervening stromal cells, which are the basic cells in this growth. The stroma is composed of spindle cells and is very, very cellular. Mitotic figures are present. We have not, however, in our experience found the mitotic index, or count, among these stromal cells in biopsy material to be helpful in any way in predicting the outcome of cases.

In some areas we see another feature, namely, the presence of rather young osteoid tissue. I would like to emphasize that in none of this osteoid tissue, in none of the giant cells, and in none of the spindle cells, are any mitoses found which are pathologic. In

no place are there giant cells of the Joe Louis or Rocky Graziano type, big owl-eyed cells which are obviously malignant. The giant cells are all osteoclasts. Now the presence of this osteoid tissue in a giant cell tumor is not too disturbing, especially in cases in which there has been a history of injury. Fractures occur through these bones, the bony mesenchyme is present, and the bone heals. We are perhaps thankful that there are tremendous numbers of the giant cells, because it helps us a little in pronouncing this tumor benign rather than malignant. You may see many osteoclasts in certain areas of osteogenic sarcomas that have undergone hemorrhage and necrosis, but when one finds them in numbers such as in this case, one begins to think of something other than an osteogenic sarcoma. Their presence in great numbers also will help us to rule out certain conditions like osteitis fibrosa cystica, fibrous dysplasia, benign chondroblastoma, and other growths that contain giant cells. My diagnosis in this particular case is benign giant cell tumor of the scapula.

By way of discussion I might say that Dr. D. C. Dahlin, my associate, has recently gone over 101 cases of giant cell tumor in which he had all the old slides and all the old tissue preserved in formalin. The clinical behavior of these tumors was not measured by the mitotic activity as shown in specimens from curettements. Ten of the cases were malignant, but in only 2 did malignancy appear as a primary change in the tissue originally examined. These 2 tumors exhibited fibrosarcoma in a neoplasm which elsewhere had all the features of giant cell tumor. The other 8 of those growths (7 fibrosarcomas and 1 osteogenic sarcoma) were from patients who had had an inadequate curettage from 4 to 20 years previously, and then had been treated with irradiation therapy. It is Dr. Dahlin's and my feeling that roentgen-ray therapy to a giant cell tumor exposes the patient to the danger of developing an irradiation induced sarcoma.

I admit that there are several cases in the literature in which a neoplasm resembling this microscopically (many giant cells and a richly vascular stroma, with no pathologic mitotic figures) has metastasized to the lungs. I saw one at the Armed Forces Institute of Pathology; I saw another referred case; and I read of a case in the *British Journal of Pathology* a number of years ago. Those cases are very, very rare, and deserving of reports in the literature as real "odd balls." No less than 10 per cent of Dr. Dahlin's original group designated as giant cell tumors proved on more adequate sampling to be osteogenic sarcoma, which brings up the important point that the student can do the amputation, but the professor should do the biopsy and obtain adequate material which should be sampled adequately by the pathologist to rule out the presence of a sarcoma with giant cell–like areas.

Sixty per cent of our series of 101 giant cell tumors were in the epiphyses of the lower extremities, and none were scapular in origin or primary in the vertebral bodies. All of our vertebral and skull tumors originally reported as giant cell tumors turned out on analysis to be examples of aneurysmal bone cysts. These can be treated and cured in almost all cases by curettage. Such treatment is inadequate for giant cell tumor of the ordinary type in a long bone.

Submitted Diagnoses.—Giant cell tumor, 27; giant cell tumor in aneurysmatic bone cyst with fibrous dysplasia, 1; osteofibroma with hemorrhage, 1; osteitis fibrosa cystica, 1; malignant giant cell tumor, 9; osteogenic sarcoma, 5; giant cell osteosarcoma, 1.

Dr. Dockerty: The diagnosis of giant cell tumor in aneurysmatic bone cyst with fibrous dysplasia is perhaps sparked up by the areas of bone and the areas of fibrosis which were present. I believe in an aneurysmal bone cyst the diagnosis is fairly easy to establish. In the first place, grossly there is a solid, meaty mass in the bone which shells out readily; along with this are tremendous, vascular spaces, so that you may have difficulty getting good sections through solid areas of tumor. Giant cells of course are present in the solid areas, but other zones have few of them. In this particular case the giant cells were scattered pretty evenly throughout. As regards osteitis fibrosa cystica, you can pick out from such a case zones in which there are many giant cells. It would be difficult, however, to duplicate the slide placed in our seminar group.

I do not believe that this is a malignant giant cell tumor, and I also do not attempt to grade such tumors.

The diagnosis of osteogenic sarcoma was probably based on the presence of osteoid, which I interpreted as being bone that formed as the result of fracture through the tumor. The scapula is a thin bone, subject to a lot of trauma, and it will break easily. As a matter of fact, it is difficult to establish in the scapula the fact that a given tumor is central in origin, because the bone in certain areas is only a millimeter or two thick. By giant cell osteosarcoma I suppose is meant an osteogenic sarcoma with numerous giant cells, and I pointed out the reason for my rejecting that particular diagnosis. The giant cells are just too numerous, and there are no pathologic mitotic figures in the stroma. My diagnosis is cellular, but benign, giant cell tumor of the scapula.

Dr. S. W. Bohls, Austin: The patient is still living and well after one year.

Dr. Leo Lowbeer, Tulsa, Okla.: Could you give us your ideas about the nature of the giant cells in giant cell tumors, and also about the relation between the giant cell tumors as we know them and those tumors which have been called with various names, such as chondroblastoma and chondromyxofibroma?

Dr. Dockerty: I am not sure I can answer your

question. I personally regard the osteoclast-like cells in giant cell tumor of bone and in fibrous dysplasia as fused spindle cells. I think that their origin is bone mesenchyme. I feel that mesenchymal cells are pluripotential and that the same cells which in this case went on and laid down osteoid also produced giant cells. We see them producing cartilage, particularly in osteitis fibrosa cystica. Chondroblastoma of Codman is a very interesting tumor. Some areas in it are fairly typical of giant cell tumor; other zones recall the picture of chondrosarcoma. You have to go back to your philosophy again and realize that perhaps both of those cells came from the same mesenchyme and that when you find the two in association, the neoplasm is benign. In our series we had several Codman tumors that were called malignant, and major surgery was done for them; yet in recent years we have been just locally excising them.

CASE 5

Diagnosis.—Grade IV chondrosarcoma of scapula.

Contributor.—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

History.—A 64 year old woman was admitted to the hospital with a huge tumor of the shoulder of 2 years' duration. At one point it extended through the skin, producing a 4 by 6 cm. area of superficial ulceration. Roentgenograms showed destruction of almost the entire scapula, and erosion of the upper end of the humerus, lateral portion of the clavicle, and two adjacent ribs. The lungs were clear, and an intravenous pyelogram was normal. At operation the tumor was essentially a single, large, well circumscribed mass. The central portion was necrotic, cystic, and hemorrhagic, but the peripheral portion was firm, gray-white, and somewhat translucent; with an ill defined lobular pattern.

Dr. Dockerty: I believe there is one roentgenogram in this case.

Colonel Harrell: Actually all that you can see on this slide is the enormous soft tissue tumor. There are some strands of calcification that appear to be in the tumor, though the bone detail is not well defined. The humerus merges with and disappears in the tumor. It is hard to see on this lantern slide the little details which we like to see; but I assume that this means that the head of the humerus is entirely destroyed. It speaks in the protocol about some ribs being destroyed; I can not make that out, either, but I assume that that would be just by pressure. The soft tissue calcification would suggest an origin in synovia or some structure about the joint, or periosteum. Occasionally, of course, cartilaginous tumors will progress to the point of calcification. Osteochondromas or chondrosarcomas will lay down calcium out in their peripheries.

Dr. Dockerty: This, incidentally, is a real Texas tumor, the size of a Texas grapefruit. We are told that it is scapular in origin, and I gladly accept that biasing information because I think that in many of these growths the pathologist needs all the help he can get. He must pay attention to the age of the patient, the location of the tumor, and so on. For instance, a basal-squamous cell cancer from the side of the face acts like a basal cell cancer. It may eat your head off eventually if you do not do something about it, but it is not going to metastasize. The same appearance shown in an anorectal cancer has serious import. So the clinical data are important. They always should be available to the pathologist, along with a generous biopsy specimen so that he may sample different areas. As we look at this neoplasm we note that it is extremely cellular; there is some hemorrhage in it, and areas of necrosis, all of which are helpful in arriving at at least one station along the way. This growth is malignant.

Basically the cells are more or less round, but unlike those of reticulum cell sarcoma many seem to be surrounded by narrow clear zones. The margins of these boundaries seem to be outlined by reticulin. Giant cells, binucleated forms, and numerous mitoses attest to a high degree of anaplasia. A few of the cells have vacuolated cytoplasm. Here and there is a bit of stainable ground substance which to me resembles chondromucin rather than osseomucin. In a desperate effort to arrive at a diagnosis in this tumor I went to the literature and also reviewed our series of 30 to 40 malignant tumors of the scapula. I found that chondrosarcoma accounted for well over half of the primary malignant tumors. That was helpful, because this might be a chondrosarcoma. Yet, as I went over our series of chondrosarcomas I did not find any that were quite as malignant as this one. It is rare to get a degree of anaplasia so marked that one can not even decide on the basic cell. Ackerman and O'Neil, in a recent article, however, do show an example of a grade IV chondrosarcoma with a comparable microscopic picture. Finally, I got out our slides on Codman's tumor, because I wanted to see what real juicy-looking cartilage cells appeared like when they were packed closely together. Codman's tumor is a benign tumor, but it does show young cartilage cells. I was struck by the resemblance to the rounded cells with the clear zones about them, and finally decided that here, perhaps, if we were dealing with a primary bone tumor, it was a high grade chondrosarcoma of the scapula.

From the microscopic picture we can easily rule out other primary bone neoplasms such as myeloma, where the plasma cells are pretty easily identified. We can also rule out by the same token Ewing's tumor, with its tiny spindle shaped Ewing-tumorcytes. (I like to call them this because I do not know where they come from.) We cannot quite rule out an osteogenic sarcoma. An osteogenic sarcoma could show this degree of malignancy, but you would expect to

find a little osteoid in it. Actually, I do not think it would make much difference to the patient whether this tumor is an osteogenic sarcoma, a rhabdomyosarcoma, or a chondrosarcoma. It is a grade IV sarcoma and the prognosis should be poor indeed. Of the soft tissue tumors, synovial cell sarcoma, rhabdomyosarcoma, and liposarcoma are most likely to confuse the issue. In synovial cell sarcomas you usually have clefts, and you will find calcification. The cells are spindle shaped, and the presence of giant cells is unusual. Rhabdomyosarcoma is sometimes difficult to diagnose, but thanks to Arthur Purdy Stout and others we have come to regard fibrosarcoma-like lesions with giant cells as being basically rhabdomyosarcoma or liposarcoma. I always like to give the patient the benefit of any doubt, and if I cannot decide whether a tumor is liposarcoma or rhabdomyosarcoma, I will put my first choice as liposarcoma because some of those respond in a miraculous way to roentgen-ray treatment.

Dr. Manhoff: The chest plate at the time of surgery was perfectly clear, but 4 weeks after surgery the chest was completely loaded with huge metastatic nodules and the patient died 3 weeks later. At autopsy there were abundant metastases almost completely replacing the lungs. There was one single nodule in the liver, one small metastatic nodule in the ileum, and abundant recurrence at the site of the original tumor.

Dr. Dockerty: That perhaps is not surprising in view of the very anaplastic nature of that lesion.

Submitted Diagnoses.—Sarcoma, 37 (reticulum cell, 7; chondrosarcoma, 7; liposarcoma, 6; undifferentiated, 5; Ewing's, 4; synovial, 2; angiosarcoma, 2; osteosarcoma, 2; rhabdomyosarcoma, 1; fibrosarcoma, 1); metastatic carcinoma, 4; miscellaneous malignant tumors, 4.

Dr. Dockerty: I tended to rule out reticulum cell sarcoma because of the clear zones around the cells and the presence of giant cells, which obviously were not Reed cells. A few folks I see did agree with me on the diagnosis of chondrosarcoma. It could be a liposarcoma, but I have seen only 1 liposarcoma in a bone. Of course a tumor of this size with involvement of bone could have been primarily a soft tissue tumor. Undifferentiated sarcoma I think is an excellent diagnosis. Ewing sarcoma I would disagree with because of the cell size. The cells in the typical Ewing's tumor are about the size of lymphocytes; they are about as closely packed, and have about as much cytoplasm around them as one sees in a lymphosarcoma. Synovial cell sarcoma I reject because of lack of spaces and the presence of giant cells. The lesion did behave like a synovial cell sarcoma, however, in metastasizing to the lungs. It could be an angiosarcoma, but I was not as impressed perhaps with the vascularity as were the two people who made this diagnosis. It certainly could be osteogenic sarcoma, but I do not think the evidence in the slide that I had was good for an out-and-out diagnosis. The presence of big straplike cells indeed suggests a diagnosis of rhabdomyosarcoma. I think that a "fibrosarcoma" with such giant cells more likely would be a liposarcoma, however. Metastatic carcinoma is a possibility I had not thought of; however, I believe that if it is a cancer, it is one which is so closely imitating a sarcoma as not to affect the treatment.

CASE 6

Diagnosis.—Primary chondrosarcoma (grade I-II) of bone (humerus).

Contributor.—Capt. R. C. Schaffer, USAF Hospital, Lackland Air Force Base, San Antonio.

History.—A 33 year old man first complained of stiffness and pain in his right hip a year before. An operation of the hip was performed, and the synovial membrane was apparently removed, although it appeared normal. Stiffness and pain continued and another operation was performed 9 months later, at which time irregular, gray fragments measuring up to 2 cm. were removed from the joint. Slides are from this operation.

Colonel Harrell: I am afraid I will not be able to contribute much from this roentgenogram. There is marked soft tissue swelling and soft tissue tumefaction around the right hip. There seems to be some bone destruction of the ilium just above the acetabulum, but the soft tissue tumor goes clear down into the thigh and the hip. It is an enormous tumor. The appearance certainly is highly suggestive of malignancy in this case, the primary site of which I would have to guess at entirely. The ilium on the medial side appears normal.

Dr. Dockerty: Would the contributor care to interpret the gross photographs in this case?

Captain Schaffer: There was a 4 cm. tumor projecting through the obturator foramen. The tumor had also grown through the acetabulum and formed a 2 cm. mass on the inner aspect of the wing of the ilium.

When hemisected the tumor was seen to extend outward from the capsular area for a distance of from 4 to 8 cm. No remaining joint capsule or synovial membrane could be found. The joint space was lined by tumor except for the central portion of the articular surface. There was partial destruction of the joint cartilage of the acetabulum and of the head of the femur.

Dr. Dockerty: While out of line a little, I think I would like to give you at this time my diagnosis, which is chondrosarcoma of the pelvic bones. Whether it started in the ilium or the femur I cannot tell, and I do not think it would make too great a difference. In our series of cases 60 per cent of chondro-

sarcomas arose from the pelvic girdle, and if the upper ends of both femurs, the proximal ends of the humeri, and chest girdle are added, the figure reaches more than 90 per cent. It is well to remember that 90 per cent of these tumors come from areas close to the trunk. I would make the diagnosis of chondrosarcoma on the original material, and certainly on the recurrence.

Microscopically, we see an obviously cartilaginous tumor. The background matrix is chondromucin, in anybody's language. We notice that for a chondroma, the lesion is just too cellular; the nuclei are plump with prominent nucleoli. There is variations in size of the cells, with some being multinucleated; and, more important, the multinucleation is easy to find. In my opinion multinucleation in a chondromatous tumor is a worrisome feature. You cannot be guided by mitotic activity because these chondromatous tumors do not have many mitotic figures, except the one that we just diagnosed, in case 5. The islands of neoplastic appearing bone I regard as being metaplastic, and more or less incidental.

Dr. Dahlin recently has gone over more than 200 chondrosarcomas of bone seen at the Mayo Clinic over a period of 50 years. These chondrosarcomas comprise some 10 per cent of our group of primary malignant neoplasms of bone, being twice as common as Ewing's tumor and half as common as osteogenic sarcoma. It has taken pathologists about 50 years to recognize that the credentials for malignancy in cartilaginous tumors are not the same that they use for other neoplasms, and as a result these tumors were underdiagnosed. Twenty-five years ago the surgeons decided that the pathologists did not know one cartilaginous tumor from another and began doing big operations for all of them, regardless of what the pathologist reported. However, the surgeons erred in one important respect. In response to the pathologists' request for more and more biopsy material they would incise these growths and "spill" them. The recurrence rate became almost 100 per cent. (It was close to that in our series when such treatment was carried out.) Originally these tumors were called cellular chondromas, and liquefying chondromas. I remember 1 liquefying chondroma in the femur of a woman which I diagnosed. I was amazed 2 years later to find that she had generated several chondromas in the uterus, and one in the urinary bladder. I began to pull in my horns and review the literature on chondromatous tumors.

In review, and using the criteria of Lichtenstein and Jaffé, I believe that these tumors are cytologically growths which can recur, will recur in a high percentage, and metastasize in 10 to 20 per cent of the cases. Only 2 per cent of malignant chondromatous tumors in our series involved the hands and the feet, which is a point to remember. One observes many cases of multiple chondromatosis of metatarsals and metacarpals, but rarely obtains, let us say, malignancy in a cartilaginous tumor on the back of the hand. Dr. Bell remarked many years ago that the further away from the heart, the lower the incidence of malignancy. Unlike Lichtenstein, in our series we did not find any recurrent chondrosarcomas which were centrally located in bone and which at the first operation were benign. They were one and all primarily malignant. Ackerman has had the same experience. Ten per cent of our series metastasized, and of course a tremendous number recurred. Our policy on these growths now is to get the professor to do the biopsy. We make a frozen section of the tumor and render an immediate diagnosis if possible, but if we cannot we will hold it over for a day. In any case, the surgeon seals up his incision carefully. During the "big" dissection he never again allows himself to approach the tumor, because of the tendency for these tumors to seed.

My diagnosis is chondrosarcoma, arising from somewhere in the pelvic girdle.

Dr. N. E. Pond, USAF Hospital, Lackland Air Force Base: At the time of autopsy, at the site of the hemipelvectomy there was a large pseudobursa completely filled with a cartilaginous growth which extended up into the soft tissue of the pelvis with two nodules on the inner aspect of the pelvis, and then extended by direct continuity up the epidural space of the spinal cord and into the brain, again by direct continuity. I opened up the whole spine. You could follow the tumor right up into the head. There was a large epidural tumor in the skull, and in the skull it also infiltrated through the dura so that there was large subdural occipital extension of this tumor. In addition it had eroded through the skull to form a large mass beneath the scalp in the occipital region. There was invasion of every single vertebra except the cervical, which I did not examine. There were noncontinuous metastases to three ribs on the left, one rib on the right, and the right temporal lobe of the brain, and both lungs were almost completely replaced by tumor.

Dr. Dockerty: I am glad that you put this case on, because it is not often that we find one of these lower grade chondrosarcomas behaving in such an unquestionably malignant fashion, and over a fairly short period of time.

Dr. Pond: One of the consultants who saw the original lesion and who is a bone and joint pathologist made the statement that in many cases in which the surgeon notes chondromatosis of the synovia, if it is confined to the synovia itself one can more or less overlook some of the disturbing histologic features, because he has rarely seen a chondrosarcoma arising from chondromatosis.

Dr. Dockerty: I know of only 2 cases of chon-

dromatosis of synovial lining from which sarcoma developed, and I am not sure that one of them is too clear cut.

Submitted Diagnoses.—Chondrosarcoma, 40; chondroma, potentially malignant, 1; osteochondromatosis, 2; benign chondroblastoma, 1; chondroma, 1.

Dr. Dockerty: The majority favor chondrosarcoma. The pathologist who made the diagnosis of chondroma, potentially malignant, was probably worried by the cellularity. The osteochondromatosis voters are pretty well outnumbered. I would argue with the diagnosis of benign chondroblastoma because there were no osteoclastic giant cells, number one; and number two, the cartilaginous cells varied a great deal in size and shape and they were not closely packed together. I wonder if this last is chordoma, or chondroma. I am surprised someone did not include chordoma, because we find chordomas in unusual locations, and they are prone to recur. They are prone to spill unless one goes widely around them. The treatment of chordomas at the Mayo Clinic consists of a collaboration of pathologists, roentgenologists, neurosurgeons, orthopedic surgeons, general surgeons, bowel surgeons, and gynecologic surgeons. They all mill around; one takes care of the nerves, the other the rectum, the other the blood vessels, and the other the bladder. We are getting good results from this combined, terrific, all-out attack on chordomas. The pathologist, however, has difficulty getting into the operating room in these cases.

General DeCoursey: Do you want to give us a grade?

Dr. Dockerty: It is a good grade I. I will grade it low because I want to impress on the group that these borderline chondrosarcomas can metastasize by the blood stream and kill rapidly. They are dangerous lesions.

Dr. Schaffer: I notice that two people thought this was osteochondromatosis. I wonder if you would comment as to whether you saw any evidence here that you thought could have put this originally in that category. It was supposed to have looked like an osteochondromatosis when it was originally operated on, and the surgeon thought they had gotten it all out.

Dr. Dockerty: I am glad that you were not confused by that biasing information. I can see what the surgeons were talking about. It had to do with the bumpy appearance on the surface, which is par for the course in chondromatous tumors; yet you note that in their report they said that the synovial lining itself appeared normal. They apparently were grasping for straws in trying to help the patient. As was mentioned, there are only 2 cases in the literature of cartilaginous malignancy which were superimposed on osteochondromatosis. I suppose, on the other hand, that when a tumor becomes as extensive as this one, it would be difficult to decide on the point of origin.

CASE 7

Diagnosis.—Parosteal osteogenic sarcoma of the humerus.
Contributor.—Brooke Army Hospital, Fort Sam Houston.
History.—A 17 year old white boy noticed limitation of motion in his left arm 6 months prior to surgery, and increase in size of his left shoulder and pain 2 months prior to surgery. Roentgenograms were suggestive of osteogenic sarcoma. The first biopsy specimen was reported as bone, fibrosis, chronic inflammation, and new bone formation. Slides are from later material.

Dr. Dockerty: In view of the original pathologic diagnosis of "bone, fibrosis, chronic inflammation, and new bone formation," the fourth assistant apparently did the biopsy in this case and the professor the forequarter amputation which was performed about 1 month later. We have some roentgenograms of the lesion.

Colonel Harrell: There is marked overgrowth of bone which is sclerotic and dense. The cortex appears to be destroyed on both sides, and at least half the bone involved, the shoulder joint itself being spared. There is a great protrusion of overgrowing osseous tissue out into the axilla. This appearance certainly suggests the sclerosing type of osteogenic sarcoma. There is some spiculation, although it is not the sunburst ray spiculation that oftentimes accompanies these tumors. Certainly the diagnosis here is highly suggestive of a malignant primary bone tumor.

The roentgenogram of the longitudinally sectioned amputated specimen shows practically the same thing that the original roentgenogram does. I might add that laminographic studies of these lesions before they are amputated give you a similar sectional appearance.

Dr. Dockerty: The gross specimen shows involvement of the parosteal zone, an encasing and ensheathing, very extensive lesion of the upper half of the shaft of the humerus.

Microscopically it can be seen that the bony proliferation in this tumor is invading the striated muscle of the upper arm. The outstanding feature is the invasion of muscle and the production of new bone in the form of rather narrow, trabecular beams which are fairly immature, or "tumor bone." They are a sort of cross between the bone that you might find in the healing of a fracture and that of the "tumor bone" that is seen in more malignant osteogenic sarcomas.

The trabeculae are long and narrow with rather tenuous anastomoses, one with another. Large irregular compartments intervene, and these are filled with elongated spindle cells in palisade. The spindle cell element is a cellular one. There is little inflammatory

infiltrate. The spindle cells seem to run parallel to the surface of the nearest bony trabeculae.

In some areas only the spindle cell element is observed, and the picture is reminiscent of fibrous dysplasia except that giant cells are few in number and the fibrous tissue is distinctly immature. Zones of myxomatous change and of cartilaginous metaplasia are much more commonly seen than in fibrous dysplasia.

Mitotic figures are seen in some of the fibroblasts, which is unusual in fibrous dysplasia; there is pleomorphism, and some prominent nucleoli.

It is important to obtain a really generous biopsy sample from one of these tumors. You want to get the bone at the growing edge; you want to get enough bone to show the arrangement of the fibroblasts, and how juicy and active they are.

We must admit that if we are going to call this a fibrosarcoma, it certainly would be an acellular one. In this case the history, the location, the typical roentgenographic appearance, the combination of the bony beams that are sort of borderline between neoplastic bone and healing bone, the appearance of the fibroblasts, and their relation to the bony trabeculae that are formed must be considered in order to arrive at the correct diagnosis.

By way of discussion I can say that

> Vice is a monster of so frightful mien,
> As to be hated needs but to be seen;
> Yet seen too oft, familiar with her face,
> We first endure, then pity, then embrace.
> —Alexander Pope.

It is just about like this in a pathologist's attitude toward this tumor. To make a diagnosis of sarcoma on such an acellular mixture of bony and fibrous elements is at first abhorrent; but after the pathologist has seen a couple of cases, and perhaps gotten stung by one of them, and has talked to his colleagues, he then begins to entertain the diagnosis of malignancy on the basis of that mixture; and finally, after he goes over a personal series, he is willing to embrace the concept and he becomes bullheaded and biased about it, even as I am now. We have seen 20 of these tumors in young people. Four of our 20 tumors exhibited no more cellularity than this case, but eventually metastasized. The original diagnosis in some of the older cases in our series was osteochondromatosis with inflammatory change—fibrous, inflammatory tissue, just as it was here; but we have learned now to listen to the roentgenologist and to look at the roentgen-ray plates ourselves and correlate the whole picture.

My diagnosis, based on the appearance here in comparison with 20 of our own, is a parosteal osteogenic sarcoma: a tumor that will recur; a tumor that will metastasize in a high percentage of cases; a tumor that has to be treated radically at the start, after having been recognized as such, and not partially excised. Such an attack gives the patient the best chance for a cure.

Submitted Diagnoses.—Fibrous dysplasia, 10; myositis ossificans, 6; osteochondroma, 5; neurofibroma, 1; ossifying fibroma, 1; osteoma, 1; bone angioma, 1; osteochondritis, reparative stage, 1; sarcoma, 15 (osteogenic, 12; fibrosarcoma, 2; parosteal, 1); osteochondroma with malignant change, 3.

Dr. Dockerty: Bone and cartilage are "par" for fibrous dysplasia; but the bone is not laid down in these little tenuous beams, and the fibroblasts are not as numerous; they do not run parallel to the trabeculae. Myositis ossificans can really cause difficulty, because in it you observe fibrous tissue that is even much more cellular than in this case; you find bone that looks like tumor bone; but again, the roentgenographic appearance is helpful because myositis ossificans begins in the muscle, not in the bone, and the tumor as seen grossly and by roentgen ray will touch the bone tangentially, but it will not encase it. This is an important difference. Incidentally, myositis ossificans is much more frequently called malignant than is this condition; so we have the paradox that the benign condition, myositis ossificans, is often called malignant, and on the other hand this parosteal osteogenic sarcoma is interpreted as a benign tumor because of the strange cellular mixture, lack of cellularity, inflammatory component, and so forth. The presence of scanty amounts of cartilage does not justify a diagnosis of osteochondroma.

Somebody thought this was a neurofibroma. I have never seen a neurofibroma primary in bone. I have tried to find one among a group of painful osteoid osteomas, but without success. If you found such a cellular composition (without bone and cartilage) in an eighth nerve tumor, you might diagnose it neurofibroma; but you certainly should worry about the presence of mitotic figures. Ossifying fibroma is not a tumor that encases the upper ends of long bones in young people. Favoring angioma was the fact that this was quite vascular; but in my experience angiomas of bone are mostly in the vertebral column or they are associated with congenital anomalies and elongation of a limb, and the vascular component is much more pronounced than it was in this case.

This looks much too tumefactive to be an osteochondritis, reparative stage. We have 15 diagnoses of a low grade neoplasm that is producing bone and a little bit of cartilage. As for osteochondroma with malignant change, I do not think there was enough evidence of cartilage in this case; and then as I mentioned before, these other criteria, namely, the character of the bone beams and their relation to the fibroblasts, are the features which are characteristic of parosteal tumors of this type.

In cases such as this the pathologist should let the

roentgenologist make the diagnosis. He should go out and have a nice day of golf, and then go back to the laboratory three or four days later and dissect out the amputated specimen, instead of deferring for a week a biopsy diagnosis such as "fibrosis, new bone, and chronic inflammation." Well, I am out on a limb now. Parosteal osteogenic sarcoma, low grade, an unusual newcomer in the field of tumor pathology.

Lt. Col. James L. Hansen, Brooke Army Hospital: The only follow-up we have is that after the forequarter amputation the patient did get along well. We do not have a late follow-up on him.

Dr. Dockerty: As I mentioned, 20 per cent of our series of 20 metastasized, and the figure in the literature is about 10 to 20 per cent. They have to be treated radically at the start, as this one was, to expect good results.

General DeCoursey: May I ask if you would be influenced if this young man had come in giving a history of having been previously hurt in, say, a football game and had developed a swelling in a muscle?

Dr. Dockerty: Well, again, I would let the roentgenologist help me.

General DeCoursey: Or would you see this same picture microscopically?

Dr. Dockerty: The point is that I do not believe you would see the same gross and microscopic picture. The bone in a myositis ossificans is more like tumor bone, and the trabeculae are irregular, with Chinese and Japanese characters; the fibroblasts do not line up as in this tumor; it is infinitely more cellular; and there is the tell-tale hemorrhage. Also, myositis ossificans, when it involves bone, involves bone by a globular mass touching the bone tangentially.

Colonel Harrell: This lesion, the calcified hematoma, as I like to refer to it more than as myositis ossificans, touches bone but does not destroy bone cortex. The actual cortex of the bone may merge into it, but it is not destroyed. I might add that fibrous dysplasia involves the cortex of the bone, but it also expands the cortex. It does not grow on the outside of it, and it does not destroy it. Your x-ray consultant can steer you clear of fibrous dysplasia and calcified hematomas pretty well.

Dr. Dockerty: I strongly recommend that you get on friendly terms with your x-ray colleague. He will even teach you something about interpretation of his plates. When you see the clinicians getting up and looking at roentgenograms and diagnosing duodenal ulcers, perhaps you should be a little bit ashamed that you cannot at least do that much.

Dr. J. H. Childers, Galveston: At least in some of the sections from four sets which we had an opportunity to review, there were little areas of osseous tissue and fibrous tissue which were covered by cartilaginous caps with uniform hyaline cartilage cells. Where did these cartilage cells come from?

Dr. Dockerty: I believe that these fibroblasts are osteogenic, and they are chondrogenic, too. The tumor is trying to differentiate into bone, and it has a cellular fibrous component which can also lay down cartilage cells over the end of the bone that they have produced. That perhaps is why the suggestion was made that this was a malignancy in an osteochondroma.

CASE 8

Diagnosis.—Bilateral metachronous mammary carcinoma and associated metastasizing liposarcoma.

Contributor.—Brooke Army Hospital, Fort Sam Houston.

History.—A 27 year old woman had a left radical mastectomy for carcinoma in December, 1950, at which time one axillary node contained tumor and she was given postoperative irradiation. In October, 1953, she had a right radical mastectomy for carcinoma, which was interpreted as a second primary tumor. In April, 1954, a tumor in the left supraclavicular region was noted. This area was again treated with roentgen ray. Further irradiation to the area was given in December, 1954, and in June, 1955. At the time of death in December, 1955, there was a large fungating and ulcerated lesion involving the left shoulder and extending through the chest wall into the lung.

Dr. Dockerty: In this case the big question is whether we are dealing with one, two, or three malignant tumors. The left breast lesion was a grade III scirrhous adenocarcinoma with involvement of one axillary lymph node. I would have liked to see more sections of this if they had been available, because it would not have surprised me to find some comedo areas. In our experience, while 4 per cent of ordinary breast cancers are nonsimultaneously bilateral, that figure is at least double when the type of growth in the first breast is a comedo, or duct, cancer.

The right breast growth which appeared almost 3 years later was, in my opinion, a grade III comedo adenocarcinoma with extension to, or rather, multicentric involvement of, many terminal acini. Stewart calls them lobular carcinomas in situ because of the thought expressed by some people that the real acini in the breast are something that pop up only during pregnancy; but even in male breasts one notes tiny buds at the ends of ducts, and in certain cancers, notably in comedocarcinoma involving large ducts, the process, being multicentric, goes out and involves the terminal buds. I could not be certain about breaching of the ductal basement membrane in this lesion and accordingly feel that the evidence is strong for the thesis that the two breast growths are independent.

The highly anaplastic spindle cell neoplasm which fungated through the soft tissues of the left shoulder, and terminally metastasized to the lungs, is different from the two cancers which preceded it. The anap-

lasia culminates in the formation of huge, sometimes vacuolated uninucleated and multinucleated giant cells. No areas of epitheliogenesis are apparent. Fat stains are weakly positive.

One per cent of our breast cancers are simultaneously bilateral, and I think that a good many of those are metastases from one side or the other, for two reasons. Most of them are medial lesions, which we know tend to spread to the other side; and only 10 per cent of the patients live 5 years, which prognosis is about four times as bad as for ordinary single cancer in one breast. Five per cent of breast cancers appear in the opposite breast within a period of 2 or 3 years, and are fairly obvious metastases, usually being associated with other evidence of dissemination. In an additional 4 per cent of cases of breast cancer (8 per cent in comedocarcinoma), however, a cancer will be found in the opposite breast within a period of 3 to 5 years, and the prognosis in that particular group is relatively so good that we know that many of them must be independent primaries rather than metastatic lesions. The second breast showing, as it did, a comedo appearance, I think is pretty good evidence that this woman had independent cancers of the breast rather than a metastasis, because were a breast cancer to metastasize from the left side to the right, it certainly would not enter and involve primarily the ducts.

As regards the soft tissue tumor, one must keep in mind that adenocarcinoma can appear as spindling growths which are apt to be mistaken for sarcomas. The oat cell carcinoma of the lung is a good example of this, and certain thyroid growths produce giant cells in addition to spindle cells. Squamous cell cancer originating in old chronic sinus tracts is perhaps the most notorious for its masquerading tendencies in this regard. In all instances, however, the primary tumor shows the "sarcomatous" metaplasia. In this case the cancers of the breasts did not display spindling tendencies. A spindle cell neoplasm, the angiosarcoma, occurs in lymphedematous areas following radical mastectomy, but the microscopic appearance of this rare neoplasm features vascular and lymph spaces lined by tumor cells.

The supraclavicular neoplasm could be a fibrosarcoma with anaplastic giant cells and some fatty degeneration, and in such a growth we would not expect to find much collagen. However, I have gradually grown into the belief fostered by Stout that such bizarre giant cells in a neoplasm otherwise resembling fibrosarcoma make the growth a liposarcoma. Some of these tumors are radiosensitive. Perhaps to persist in the older notion that the growths are fibrosarcoma, and notoriously radioresistant, is to deny the patient a useful form of treatment.

I was tempted to implicate roentgen-ray treatment in the genesis of the supraclavicular tumor, but decided that the evidence was too inconclusive unless I was shown that the growth originated in bone. The carcinogenic action of ionizing rays is well documented for osseous tissue, but even here the latent period is longer than in this case.

I think this unfortuante girl had two independent cancers of the breast and, for some unknown reason, developed a third cancer—liposarcoma.

Submitted Diagnoses.—Rhabdomyosarcoma, 22; sarcoma, 16 (fibrosarcoma, 8; liposarcoma, 4; post-irradiation, 2; osteogenic, 1); metastatic cystosarcoma phyllodes, 1; metastatic carcinoma, 5; malignant giant cell tumor, 1.

Dr. Dockerty: I am not ruling out rhabdomyosarcoma, and I would like to point out that when you have a grade IV sarcoma and you do not know what it is, be frank and say so. The same might be said of fibrosarcoma and liposarcoma, but I favor the latter, which will let the patient have the possible benefit of some roentgen-ray therapy. I have already given my views that there was probably no relation between this tumor and irradiation, and that was probably what the person who diagnosed osteogenic sarcoma had in mind. In previous cases we covered cystosarcoma phyllodes and giant cell tumors. As concerns metastatic carcinoma, I do not believe that this is the correct diagnosis, but I could not rule it out without studying thousands of sections on the case.

Dr. N. E. Pond, USAF Hospital, Lackland Air Force Base: How do you like the diagnosis of malignant mesenchymoma?

Dr. Dockerty: That is about an $8 word, and in these times of inflation I suppose that one perhaps should use it more often.

Dr. J. P. Ferguson, Shreveport, La.: In view of this 8 per cent bilaterality, I wonder if you have a comedocarcinoma on one side, could you justify prophylactic simple mastectomy on the other side for reasons other than making the patient symmetrical?

Dr. Dockerty: That is an interesting question, and fortunately for us sometimes the patient decides the issue because she gets lopsided and wants the other breast removed. I would not recommend it.

CASE 9

Diagnosis.—Acinic cell carcinoma of the tongue.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 49 year old woman had a recently growing hard mass the size of a small pecan near the base on the right side of the tongue, near the inferior pole of the right lingual tonsil, and involving the right pharyngeal wall. Resected portions of the tumor showed a gray, moist, slimy cut surface.

Dr. Dockerty: I thought I was going to have a breather on this case until I noted that the neoplasm

was covered with squamous epithelium and that it involved the tongue.

The cells composing the tumor are basically large clear cells with eccentric, dark staining nuclei. They are closely applied one to another in clusters. Here and there throughout the tumor, merging with the clear cells, are cells in which the cytoplasm is stainable and granular. The granules are fine and somewhat basophilic. These granules have been interpreted as being zymogenic by those who believe that the tumor arises from acinar elements of salivary glands. About 10 per cent of the tumors exhibit large cells with a cytoplasmic content of even larger granules, and occasional tumors contain cells of all three types.

The stroma is scanty, and one does not encounter in it the cartilaginous material of a mixed tumor nor the hyaline ground substance of a cylindroma. The cells are not supposed to be mucicarminophilic as in mucoepidermoid tumors, but I have seen one exception to this rule. These tumors do occasionally have mitoses.

My diagnosis in this case is acinic cell carcinoma of the tongue, a relative newcomer in the field of oncology. These tumors were originally described in 1924 by Masson; called parathyroid rest tumors in 1929 by Frannsen; termed salivary glandular adenomas by Godwin and Colvin in 1948; and finally settled once and for all as a tumor sui generis in 1953 by Foote and Frazell, who reported a series of 21 cases. Three of their 21 patients died of the disease, 2 with distant metastases, and the recurrence rate was very high. Not enough of these tumors have been reported in the literature as yet to decide upon criteria for malignancy, but some can and do metastasize. I would not like to say whether this one is capable of metastasizing or not; it is not particularly cellular, however, and mitotic figures are rare.

To my knowledge no such tumor has been reported as being primary in the tongue, but this neither alarms nor surprises me. Most investigators have not yet gotten around to reporting their parotid examples, simply because recognition of the tumor as a distinct type has been so recent. It is my firm conviction that in due time acinar cell cancers will be reported as arising from the submaxillary and lachrymal glands; the floor and roof of the mouth; and the pharynx, nasopharynx, trachea, and esophagus. Possibly the dermatologist also will discover that some of their poorly understood tumors of sweat glands belong to this selfsame category.

Submitted Diagnoses.—Acinic cell carcinoma, 11; adenocarcinoma, 8; mucoepidermoid carcinoma, 5; metastatic carcinoma from kidney, 4; mucin producing carcinoma, 1; adenoma of salivary gland, 8; mixed tumor of salivary gland, 3; lymphosarcoma, 1; chemodectoma, 1; lymphoepithelioma, 1; chordoma, 1; craniopharyngioma, 1.

Dr. Dockerty: Quite a number agree with my diagnosis and that includes, I suppose, at least some of those who did not specify the type of carcinoma. This tumor did not exhibit the epidermoid features of a mucoepidermoid carcinoma, but these lesions can contain mucus. Up to yesterday I knew of 1 acinic cell tumor that had mucus in it, and yesterday I was shown another that contained plenty of mucus. As a result, we are going to have trouble differentiating some of these tumors, but I do not suppose that it would make a great deal of difference if you made the mistake of calling an acinic cell tumor a mucoepidermoid carcinoma, because they have about the same degree of clinical malignancy.

The diagnosis of metastatic carcinoma from the kidney is probably based on the clear appearance of the cells; and as a matter of fact, as far as the granular cells are concerned, at least 2 per cent of hypernephromas are purely granular, and an additional 10 per cent will show some granular cells. It would be unusual, however, to have a solid hypernephroma metastasize to the tongue. You might consider a mucinous carcinoma, and here we have such a diagnosis. The nuclei get pushed off to one side in the clear cells, giving them a signet ring appearance; however, stains for mucus on this particular tumor were negative. There are some salivary gland adenomas in the literature, but this looks too unlike salivary gland for me to accept such a diagnosis. The oncocytomas are granular cell tumors, perfectly benign, and they sometimes contain tiny ducts. Although there were some salivary ducts in this particular neoplasm of the tongue, they were normal included ducts, and were not the product of the tumor. The absence of a cartilaginous-like stroma and ductlike structures pretty well rules out a mixed tumor. I believe the cells in this tumor are too large and too clear for a lymphosarcoma. As for chemodectoma, such tumors are much more vascular than this one; the granular cells, which are important in making the diagnosis, are much larger and they vary tremendously in size; and the tumor has a distinctive lobular architecture wherever you see it. I do not think this could be a lymphoepithelioma; this particular tumor had too many clear cells for me to entertain that particular notion. I am glad to see the diagnosis of chordoma. Chordomas have clear cells and granular cells; but whereas this tumor grew in tremendous sheets, the chordoma tends to run out in little cords and islands. One is likely to mistake a chordoma for a mucinous cancer on the one hand, or a chondrosarcoma on the other. The tongue would be an unusual place for a craniopharyngioma. You should be able to identify squamous epithelium, basal epithelium, and stellate reticulum; and those features are not present here.

Dr. A. O. Severance, San Antonio: We did mucicarmine stains on this because of those clear cells,

and if you look long and hard, an occasional cell stains with mucicarmine, and a rare glandlike space will have some pink material in it. Secondly, this patient received roentgen-ray therapy, and now, seen this fall (1956), some 16 months after excision, was still well and without evidence of recurrence or metastasis.

Dr. Leo Lowbeer, Tulsa, Okla.: Did these cells contain lipids?

Dr. Dockerty: Not to my knowledge.

Lt. Col. J. H. Hansen, Brooke Army Hospital: We also saw a recent case of the same type of tumor—acinic cell carcinoma—which was in a minor salivary gland of the hypopharynx, and our fat stains on that were negative.

General DeCoursey: Except for this case how many people have seen acinic cell tumors outside of the parotid? Only one!

Dr. J. B. Hutcheson, Dallas: This other was one of Dr. Charles Martin's cases in Dallas, and the acinic cell lesion arose in one of the accessory salivary gland deposits in the posterior pharynx.

General DeCoursey: May I ask, does this bring up any discussion on the philosophy that a pathologist looking through the microscope histologically cannot tell whether a tumor is benign or malignant? All he can do is identify the picture, and then by knowing the natural history of this picture, say what is going to happen.

Dr. Dockerty: That is right. Virchow, looking through his microscope and seeing his first chondrosarcoma, without knowing the clinical history could not say what that tumor would do. But pathologists learn from experience. A given microscopic picture usually will produce a certain outcome, and I think that within 10 years we perhaps will be able to look through the microscope and decide which of these acinic cell tumors are going to metastasize and which ones are not.

CASE 10

Diagnosis.—Angiolipoleiomyomas (multiple) of kidney.
Contributor.—Dr. Dockerty.
History.—A 29 year old woman had a mass in the right side for 6 months, with fairly rapid increase in size and some tenderness for 2 months. Pyelography disclosed a mass involving the upper pole of the right kidney.

Dr. Dockerty: Gross features of interest in this kidney included the occurrence of no less than 6 yellowish brown, encapsulated masses, the largest measuring 10 cm. in diameter and the remainder varying from 2 cm. to 5 mm. The patient did not have tuberous sclerosis.

The microscopic high lights feature a mixture of embryonic smooth muscle cells, fat cells, and the presence of numerous and thick walled blood vessels. These vessels constitute the most peculiar feature of this lesion, as they have the caliber and wall thickness of arteries, yet they are different in that special stains reveal absence of elastica. Around them and disposed in a semiconcentric fashion are aggregates of smooth muscle cells which appear to be young. The third component, namely, the adipose connective tissue, likewise is peculiar in that it exhibits a range of cytology varying all the way from embryonic lipocytes to fully mature fat cells. The cellularity of the entire mixture is such that malignancy is strongly suspected, and without a doubt many of these cases have been hidden away under such diagnoses as liposarcoma and mixed tumor. Mitotic figures, paradoxically enough, are hard to find. Periodic acid-Schiff stains show occasional pink staining granules in the spindle cell elements. The growth is an angiolipoleiomyoma.

Jacobeus in 1905 first noted the association of sizeable renal "capsulomas" and tuberous sclerosis. His capsulomas were remarkable in that they contained thick walled vessels. Gordon in 1939 first noted that some tumors of this type could attain quite a large size and require surgical attention. To my knowledge there are about 8 similar tumors in the literature. Several have been called malignant, although none have metastasized. The tumor in Berg's patient was interesting in two respects: (1) it had invaded 8 tributaries of the renal vein and (2) it was associated with metastasis from a bronchogenic carcinoma. I have seen 2 of these tumors in our laboratory and have reviewed 1 additional case in consultation. They are about the only large benign growths seen in the kidney. They have a microscopic appearance identical to that seen in the tiny capsulomas of the necropsy pathologist and the intermediate sized lesions that have interested the neuropathologists because of an association with tuberous sclerosis.

Are they vascular in origin? Are they hamartomas? Are they neuroectodermal and basically vascular neurilemomas? The answers to these questions are not known.

Submitted Diagnoses.—Angiomyolipoma, 13; lipoma, 2; hemangioendothelioma, 3; hemangiopericytoma, 3; paraganglioma, 2; liposarcoma, 7; fibroliposarcoma, 1; vascular leiomyosarcoma, 1; angiosarcoma, 1; malignant angiomyolipoma, 2; mesenchymoma, 6; Wilms' tumor, 2; carcinoma of suprarenal, 1.

Dr. Dockerty: Angiomyolipoma is perfectly acceptable. My argument against lipoma is that this is too cellular. Watch out for cellularity, however, in retroperitoneal lipomatous tumors and in lipomas that occur around the pelvic girdle and the chest and upper arm. The diagnosis of hemangioendothelioma was undoubtedly brought about by the observation that this is a very, very vascular tumor. Against such

a diagnosis is the fact that these blood vessel walls are so tremendously thickened. Then, of course, you have to explain away the fat, and in addition the pericytes. Hemangiopericytomas do not have a fat component, whereas these tumors are encapsulated and the fat is an integral part of the neoplasm; it is not just something that has been invaded. The vascularity would suggest a paraganglioma and some of the pericytes were larger than others, but you do not get thick walled vessels devoid of elastica in paragangliomas. Liposarcoma has been the commonest error in diagnosis in our series, and fibroliposarcoma is closely related. With the diagnosis of vascular leiomyosarcoma you again encounter the difficulty of not accounting for the presence of fat as an integral part of the growth. As for angiosarcoma and malignant angiomyolipoma, we have to face the fact that in the literature none of these tumors have metastasized. Mesenchymoma is a good enough diagnosis, except that if one saw a mesenchymoma in the thigh it would not look like this at all. This is a tumor that has a specific cellular mixture. It is found in one location, namely, the kidney, and tuberous sclerosis is frequently associated with this lesion, so let us identify it in its own right, in some way. Wilms' tumors also are mixed tumors; they occur in the kidney; they occur in young people for the most part; they can occur in adults; but the composition is quite different. Striated muscle is found in 50 per cent of Wilms' tumors, if you look hard enough, but there was none in this. The composition in Wilms' tumor of very, very small cells that look carcinomatous but which form tubules, gradually merging off into a spindle cell, obviously malignant stroma, is something that is quite different from what we see in this particular case, so for that reason I will rule out a Wilms' tumor. Moreover, this was a multicentric growth. Carcinoma of the suprarenal I think was based on mistaking the signet ring fat cells for adrenocortical cells, and the diagnosis is negated by the finding of the peculiar blood vessels and the fat.

Dr. H. C. Allen, St. Louis: How many of those you have seen have been bilateral? We had one recently in which the lesions got very huge, with nothing that could be palpated on the other side. The kidney was sacrificed in removing the tumor, and then within the year the patient came back with huge masses on the other side. A second operation was done, removing as much of the tumor on this side as possible.

Dr. Dockerty: That is interesting. I have never seen one that was bilateral. I understand that in tuberous sclerosis the little tumors are frequently bilateral, but do not require surgery. I think you really have one for the books—a bilateral surgical angiolipoleiomyoma.

Dr. J. B. Hutcheson, Dallas: I would like to get together with Dr. Allen on a case report. We have also had a case that was bilateral. It was in a 56 year old woman who had a massive tumor, principally lipomatous, that was impinging on the renal pelvis. She must have had 12 or 13 smaller ones in the cortex. Although it was realized that she had a deformity of her opposite side, a nephrectomy was done. About 3 months later the same thing was done as in Dr. Allen's case. Her case was reported at a urological society meeting in Mexico last week, and she is presently doing fine.

Dr. Dockerty: That is truly interesting, and it would not surprise me from what you say, now, if many of these surgical cases prove to be bilateral eventually. Surgeons are not prone to take out both kidneys to let us know whether or not they are bilateral, and it is only by reporting instances like these that you can point up the fact that they may be. It is likewise necessary to emphasize the nonmetastasizing characteristics of these growths and urge surgeons to excise them locally. There was a lot of good kidney tissue in the case presented today, and the surgeon could have skimmed off the bigger lesion. He would have left 6 others, but those might have taken 30 years to give the patient trouble.

CASE 11

Diagnosis.—Hypoglycemic tumor of kidney.
Contributor.—Dr. Dockerty.
History.—A 48 year old man had experienced hypoglycemic episodes for 4 months, with weakness and sweating relieved by taking orange juice. Operation for islet cell tumor revealed no pancreatic nodules, but an encapsulated mass 10 cm. in diameter was found involving the left kidney. Postoperative blood sugar levels were normal.

Dr. Dockerty: The tumor was a large, solid, and sharply circumscribed neoplasm that may have started in the kidney, or perhaps was just compressing it.

Two features of this tumor are outstanding microscopically. First are the spindle cells, which here and there are aggregated into bundles, and elsewhere appear to be closely applied to thin walled vascular spaces. Nuclei are oval and not particularly hyperchromatic. Nucleoli are small and mitoses are infrequent, averaging one in every six or seven high power fields.

The second feature is the presence of balls and irregular star shaped masses of brightly eosinophilic material. These masses occasionally include individual degenerating cells. Amyloid stains are negative, and Masson trichrome stains suggest that the material consists of compressed collagen fibers.

Nine tumors of this type are in the literature, and up until we received the current one in our laboratory we had a series of 1 case. Our first patient had profound hypoglycemia, and her tumor was a huge

retrohepatic mass which I diagnosed as fibrosarcoma. No primary growth was present in the pancreas. At necropsy 2 years later this patient exhibited lung metastasis. The most recent report on the condition is one by Capt. Richard Silvis and Lt. Daniel Simon of Oakland, Calif. Most of the tumors have been in males, and ages have ranged from 16 to 70 years, with the majority falling into the 45 to 60 year bracket.

The essential points to remember about these growths concern first their large size—6 inches or more on an average. Islet cell adenomas productive of hyperinsulinism are measured in millimeters rather than inches. Functioning islet cell cancers are larger than their benign counterparts, but still fairly small. Second, they are all extrapancreatic. Several have been located in the chest, 1 of our tumors was in the liver, and the remainder have occurred as retroperitoneal growths, often attached to one of the kidneys. Third, nobody has extracted insulin from the growths to date. Fourth, the tumors are all fibrogenic and vary in cellularity. Some are histologically malignant and they can metastasize. In the case reported by Silvis and Simon the growth invaded the spinal cord. Finally, nobody has any clear conception as to why these tumors induce hypoglycemia. The observation of Evans that cutting the sympathetic nerve supply to the livers of cats produces low blood sugar levels may or may not be significant. These tumors are large enough to press on a lot of organs, but on the other hand so do uterine fibroids and other tumors which are not associated with this peculiar clinical syndrome.

Submitted Diagnoses.—Sarcomas, 11; islet cell tumor, 5; malignant islet cell tumor, 3; carcinoma, 1; spindle cell carcinoma, 1; adrenal cortical carcinoma, 1; hemangiopericytoma, 8; pheochromocytoma, 3; miscellaneous benign tumors, 6; miscellaneous malignant tumors, 3.

Dr. Dockerty: Sarcoma is a safe diagnosis for these tumors; they act as low grade fibrogenic sarcomas, and about one-third of the cases in the literature have metastasized. Malignant islet cell tumors that I have seen have not resembled a fibrogenic lesion at all. In our experience 80 per cent of islet cell tumors are benign and small, measured in millimeters rather than centimeters, and exhibit no mitotic activity; 10 per cent of them are intermediate in size, measuring 3 or 4 cm., and exhibit enough mitotic activity for me to call them grade I islet cell cancers. I have not seen any of these metastasize. Another 10 per cent of islet cell tumors are obviously malignant and often metastasize to the liver by the blood stream; but they are all epithelial tumors, whereas this is a spindle cell growth. I do not see enough clear cells in this case to suggest adrenal cortical carcinoma. This lesion is not vascular enough, has too much fibrosis, and is too lacking in arrangement around blood vessels to be a hemangiopericytoma. Pheochromocytomas can be pretty fibrogenic, but in my experience you will practically always find marked vascularity and great big granular cells like those you see in granular cell myoblastomas; you will also sometimes find some pigment; and the tumor is obviously much more anaplastic than this. I do not know what is included in the miscellaneous groups, but you can call this, if you want to, a miscellaneous benign fibrogenic tumor, possibly malignant, inducing, for some unknown reason, clinical severe hypoglycemia.

General DeCoursey: I wonder if Dr. Goldzieher has had any thoughts on this case or any experience with any similar cases. Here was a tumor of the kidney that caused hypoglycemia, but on chemical examination did not contain insulin.

Dr. J. W. Goldzieher, San Antonio: I take it this tumor did not destroy enough of the liver to interfere with carbohydrate metabolism.

Dr. Dockerty: No, this was a neoplasm that looked like a low grade fibrosarcoma. We have had the only case that was retrohepatic and that might have interfered with the function of the liver in any way.

Dr. Goldzieher: I certainly have nothing to add, but I would say that the inability to demonstrate the presence of insulin or a hypoglycemic substance would not be conclusive in my mind, because even with all the work that has been done on trying to demonstrate insulin in the blood, this is still an unacceptable procedure to most endocrinologists. There could well have been insulin in the tumor, and it just was not gotten out.

Dr. Dockerty: I suspect, also, that the pathologists got these insulin determinations as afterthoughts, after the tumor had been in formaldehyde for several days. It happened in our case.

CASE 12

Diagnosis.—Chronic thyroiditis, mixed type.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 64 year old woman was admitted to the hospital because of a hard swelling in her throat of 3 to 4 months' duration, and an associated choking sensation of 1 month's duration. The palpable mass involved both sides of the thyroid gland. At surgery a walnut-sized mass was found above the thyroid gland in the region of the thyroglossal duct. Both lobes of the thyroid and the isthmus were rock hard and were removed. The histologic picture of the thyroid and the mass above were similar. The patient is still alive with a mass in her neck 1½ years after surgery.

Dr. Dockerty: The microscopic alterations in this case concern both the epithelium and the stroma. The former, here and there, appears still to be in the business of producing colloid, but even in these zones there is enlargement and granularity of the cells

which have the hallmarks of Hürthle cells. The acini are separated by lymphocytic clusters; acinar boundaries are disrupted; and giant cells, probably consisting of fused histiocytes, are found enmeshed in the colloid substance.

Elsewhere the epithelial elements are much distorted and they are disposed singly, in small alveolar clusters, and in the form of short continuous files. In spite of this disorganization, however, mitotic figures are absent and nucleoli remain small.

The stromal change comprises lymphocytic infiltration on the one hand and fibrosis and hyalinization on the other. A scattering of plasma cells is also present. The presence of giant cells completes the bizarre picture. Of most importance, no papillary formation is seen. My diagnosis is thyroiditis, predominantly lymphocytic, but with a prominent fibrous component. One cannot always nicely pigeon-hole many of these cases, however.

The pathologist is "on the spot" in deciding upon a diagnosis in these atypical thyroid pictures. Fibrous thyroiditis often invades beyond the capsule of the gland, giving the surgeon a strong impression that an inoperable malignancy is present. His biopsy specimen is not likely to be generous. Microscopically this lesion, although benign, features such things as giant cells, Hürthle cells, single filing, spindling, and other signs which elsewhere would be strong evidence favoring malignancy. Struma lymphomatosa may be confused similarly with lymphosarcoma. Faced with such a diagnostic dilemma the pathologist may derive consolation (and help) from the fact that carcinoma is rare in thyroid glands which show a basic picture of chronic thyroiditis, and carcinoma is practically always circumscribed. The literature contains only 4 examples of a true intermingling of the two processes, and in these instances the papillary nature of the carcinoma made the diagnosis easy. Lymphosarcoma of the thyroid, likewise quite rare, displays a "solid" cellularity with cytologically immature cells showing little admixture with epithelium. Although found occasionally with struma lymphomatosa, the benign and malignant cell mixture is rarely so intimate as to make "occult" the sarcomatous component.

Dr. A. O. Severance, San Antonio: This was my case, and when the little nodule that was in the midline above the thyroid was received for frozen section, I thought it should be metastatic since it was not in the thyroid. The rest of the thyroid was removed and it all showed the same picture as the little nodule. Then I backtracked and called it chronic thyroiditis, but that word "cancer" had been uttered there at the time of the frozen section, so I was asked to send the slides off for consultation. We sent them up to Dr. A. P. Stout, and he upset the applecart by saying "carcinoma"; so the patient then received some deep roentgen-ray therapy. I submitted the case to Dr. W. A. Meissner, who was to give a seminar on tumors of the neck in the fall of 1955 in Chicago, thinking this one would be a good one for him. A month or so later he wrote back saying it was a very interesting case, but he did not think that the answer could be found and he did not like to put it in the seminar. He said that his personal opinion at that time was chronic thyroiditis, and he had shown it to Dr. Klinck at the Armed Forces Institute of Pathology, who had agreed. At the moment this patient is still alive and well without evidence of disease.

Dr. Dockerty: The surgeon can be very helpful to the pathologist in these cases. For instance, if you get a node definitely separated from the thyroid gland, especially laterally, which has even benign-looking thyroid cells in it, you are in duty bound to call it cancer with a primary in the thyroid gland. In the end you should get the corresponding lobe of the thyroid. Apparently in these thyroiditis cases, however, some of these nodules can be lightly attached to the thyroid and actually what you are looking at is not a lymph node with thyroid tissue in it, but thyroid tissue with lymphocytes in it.

Submitted Diagnoses.—Chronic thyroiditis, 18; chronic thyroiditis, Riedel's type, 11; chronic thyroiditis, Hashimoto's type, 5; subacute thyroiditis, 4; follicular carcinoma, 3; giant cell carcinoma, 1; Hürthle cell carcinoma with marked thyroiditis, 1.

Dr. Dockerty: We have 34 out of 40 who regarded this as some form of thyroiditis, and I am happy to see that there was a "mixture"; some called it Riedel's, some called it Hashimoto's, and some were real honest and said it was thyroiditis. In subacute thyroiditis I am more inclined to look for giant cells and a granulomatous reaction. It appears that when colloid of the thyroid gland gets released as a result of inflammation, it acts as a foreign body and will be engulfed by histiocytes. Some people say that the giant cells in that condition are just fused epithelial cells, but I never could quite accept that thesis. I think they are histiocytes that have fused together and come to resemble epithelial cells. Follicular carcinoma points up the difference of opinion among consultants to whom this case has been submitted. I do not believe I would call this a giant cell carcinoma. A number of years ago Dr. Broders at our institution used to see a lot of "strange" thyroids in patients who were treated with thiouracil. In fact, in the early days we called some of these cancer; but they did not behave like cancer. Dr. Broders wrote a paper on the change years ago in which he named it regenerative hyperplasia of the thyroid to contrast it with the hyperplasia of an exophthalmic goiter, in which there are also quite a few lymphocytes, and one can see giant cells, single filing of epithelial cells, and other credentials which in other lesions would

mean anaplasia as in cancer. These cases showed Hürthle cells that ran in cords, and big giant-like cells, but no pathologic mitotic figures. I suppose in thyroiditis you can get a similar interference with function and alterations in the follicular cells. Hürthle cell carcinoma with marked thyroiditis was probably suggeseted by the numerous Hürthle cells, but as I mentioned before, I believe when you are unfortunate enough to get cancer or sarcoma associated with fibrous or follicular thyroiditis, the cancer or the sarcoma usually will be a distinct lesion.

CASE 13

Diagnosis.—Grade II leiomyosarcoma of lung.
Contributor.—Brooke Army Hospital, Fort Sam Houston.
History.—A 23 year old white man had a history of right lower chest and upper abdominal pain for approximately 3 years. A mass in the lower lobe of the right lung was seen on routine roentgenogram 4 months prior to surgery.

Colonel Harrell: This mass is fairly round. The margins of it are rather sharp, and it does not seem to be invading. Actually, from this film, I do not think one can tell whether it is in the middle or lower lobe. There is no rib involvement to indicate metastasis, nor are there any other lesions or nodes that I can see in the chest. The best probabilities at 23 years of age are that this is inflammatory instead of a malignancy, unless it is a metastatic lesion. I like to study these lesions by laminography, for if they are malignant they oftentimes show umbilications in their peripheries where the blood vessels which supply the lesion enter. I have picked up a few that way, which made me feel rather confident that the lesion was metastatic, or at least malignant, because benign tumors usually do not demonstrate this umbilication. Another thing that laminography shows, which sometimes is not shown on the plain film, is whether or not these lesions contain calcium, and I think this also is a good differential point. As far as I can tell by looking at the reproduction, there is no calcium in this lesion.

Dr. Dockerty: This growth is in the middle of the lobe. It is fairly solid, and does not have the caseous appearance that we expect in tuberculomas. On cut surface we see that although it displaces a bronchus it does not actually ulcerate the mucosa. The washings in this case perhaps would be negative. With a circumscribed growth in the lung that looks like a neoplasm and with the age and all we would suspect that this might be metastatic.

Basically this very cellular neoplasm is comprised of plump spindle cells with elongated oval nuclei and pink staining cytoplasm which sometimes has a granular appearance. There is a definite tendency for cell grouping into fasicles and bundles which decussate one with another. Here and there are a few bands of brightly stained hyaline material.

Vascularity is pronounced, but there appear to be no vasculoinvasive tendencies even though some of the vessels are very thin walled. Malignancy is indicated by fairly marked mitotic activity and by the presence of infarction which seems to be on the basis of the tumor outgrowing its blood supply. Peripherally there are fibrosis and hyalinization which may be related to ancient infarction. Masson's trichrome stain gives a red appearance consistent with young fibrous tissue or muscle.

To recapitulate, then, one is impressed by the fact that we have here a spindle cell tumor with rather granular, eosinophilic cytoplasm; a definite tendency to form whorls, or bundles, or fasicles, that interlock one with another; fairly pronounced vascularity; and no cleft formation. My diagnosis, right or wrong, in this case is leiomyosarcoma, grade II, of the lung, possibly coming from the musculature of a bronchus.

The lung is a derivative of the primitive gastrointestinal tract, which frequently harbors myomatous tumors, benign and malignant. In the esophagus about 20 per cent will show malignant change, and this will reach 30 to 35 per cent in the stomach and about 50 per cent in the small intestine. They will not go directly to lymph nodes, but having breached the peritoneum, they will implant. When they do reach the lymph nodes, they usually do so via the blood stream from metastatic pulmonary lesions. According to Watson and Anlyan, there are 10 references in the literature on individual case reports of leiomyosarcoma (at least called that) present in the lung. Interestingly enough, the patients were all young persons. In other reported articles there are 10 references to benign neoplasms of smooth muscle in the lung; so apparently in the lung, as in the gastrointestinal tract, there is a great tendency for myomatous tumors to be malignant, a change which we expect in only about .5 per cent of the ordinary uterine fibroids. According to Evans, who I think did the best work on these lesions, the safest criterion for diagnosing malignancy in a smooth muscle tumor is the presence of more than 1 mitotic figure in 10 ordinary high, dry fields. One must be very careful about calling benign any smooth muscle tumor in the uterus or gastrointestinal tract which exhibits mitotic figures that are not too hard to find. One would suspect in this case that the tumor was metastatic. The commonest example of myosarcoma in the lung is metastasis from malignant uterine fibroids; but this was a male rather than a female, and we do not have any evidence in this case from other studies that there was, in fact, a primary tumor anywhere in the body other than in the lung. It has been strongly suggested that this lesion might be a metastatic synovioma; however, in a review of the litera-

ture, I have not found one synovioma presenting lung metastasis in the absence of a recognized primary growth. The lack of alveolar clefts and of calcification, and the relatively large size of the tumor cells further incline me away from this diagnosis.

Malignant mesothelioma was suggested by one of my colleagues. In our own group of 24 fibrous mesotheliomas, 6 were intrapulmonary, but in the region of an interlobar fissure. This lesion involved the basilar segment of the right lower lobe. Tumors of this type show clefts, they are more fibrogenic than myogenic, and they frequently exhibit a papillary pattern. I compared the sections on this case with sections of our malignant mesotheliomas of the pleura, and the resemblance was not striking.

I came to the conclusion that this was leiomyosarcoma, primary in the lung, a very rare lesion. These low grade leiomyosarcomas in the gut, uterus, and elsewhere are not too prone to metastasize; and yet one must follow the patients for 10 years or more.

Submitted Diagnoses.—Leiomyosarcoma, 7; mesothelioma, 13 (benign 9, malignant 4); fibrosarcoma, 5; bronchogenic carcinoma, 5; osteogenic sarcoma, 3; melanoma, 1; metastatic sarcoma, 1; neuroblastoma, 1; hemangiopericytoma, 5; bronchial adenoma, 2.

Dr. Dockerty: We have discussed leiomyosarcoma and mesothelioma, and I think that these are the two main choices. I believe that a fibrosarcoma of this low order of malignancy would have plenty of collagen formation so that it would not be confused with myogenic cytoplasm. A bronchogenic carcinoma imitating sarcoma would be more anaplastic, with more mitotic figures, and would not form fasicles or bundles. An osteogenic sarcoma would be rare, but I suppose possible; however, I did not recognize any osteoid or bone. We should think of a malignant melanoma, as they may spindle out and they can be amelanotic. Metastatic sarcoma is certainly a possibility. A malignant tumor from the gut, however, usually would have to filter through the liver, and from there spread to the lung. By that time the diagnosis would have been made because the patient probably would have an intestinal obstruction. Neuroblastoma is probably suggested by the peculiar palisading of the cells; but that is a characteristic of smooth muscle tumors of the gastrointestinal tract. As concerns hemangiopericytoma, I don't believe even Dr. Stout would champion the diagnosis in this case. Bronchial adenoma I think is ruled out because those tumors are pretty much recognizable as epithelial, and this was more of a spindle cell growth.

Follow-Up.—Approximately 11 months following the original surgery a second thoracotomy was performed, with the removal of a 1.5 cm. subpleural lesion from the upper lobe of the right lung showing a picture similar to that of the original tumor, and interpreted at the time as being an implant. Roentgenographic follow-up at 18 months revealed multiple asymptomatic lesions throughout the entire right lung field.

CASE 14

Diagnosis.—Choriocarcinoma of the uterus.

Contributor.—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

History.—A 31 year old white woman passed a large hydatidiform mole. Two months later a vaginal nodule was subjected to biopsy, following which a panhysterectomy was performed. The uterus showed a bulging 4 by 2 by 1.5 cm. friable mass, and there was a 5 cm. corpus luteum cyst of the left ovary. Two months after hysterectomy another vaginal nodule was excised. The vaginal nodules and uterine mass all showed the same histologic picture. The patient was living and well 2 years after hysterectomy.

Dr. Dockerty: Grossly the picture of the original lesion is what anyone would call a hydatidiform mole. "Grapes" are present in great abundance. The microscopic sections exhibit well formed villi with focal hyperplasia of the mantling trophoblasts, both syncytial and Langhan's. Like Hertig, I think that these more active moles are the ones most likely to proceed into choriocarcinoma. In any given specimen, however, regardless of location, I lean strongly away from a diagnosis of chorionic malignancy when even one identifiable villus is present. Novak, Hertig, and others concur in this opinion.

The uterus showed a red hemorrhagic nodule, but it is perhaps significant that this nodule does not extend entirely through the uterine wall.

The vaginal nodules and the hysterectomy specimen show marked anaplasia of trophoblasts, invasion of veins, necrosis of muscle, and much hemorrhage. These features, however, do not suffice for a diagnosis of choriocarcinoma unless villi are absent, and many sections must be taken to prove their absence. It has been my experience with that of many others that villi can become deported to the vagina and even to the lung. To my knowledge there are only 2 cases in the literature in which death dealing pulmonary metastases contained villi. Novak noted only 1 instance in his 74 cases of true choriocarcinoma.

No villi were present in the uterine and vaginal nodules, but this does not certify the diagnosis of choriocarcinoma. The sampling must be generous, and the finding of one villus should negate a malignant diagnosis. This must be our philosophy if we are to get a proper perspective on treatment and prognosis. Currently we are studying 3 cases with these borderline lesions known as metastasizing chorioadenoma destruens, associated with lung shadows. Each of the primary growths exhibited occasional villi in the bad company of very malignant appearing cells. These lung shadows are disappearing under roentgen-ray therapy and it is the experience of some that they disappear without any treatment. We

persuaded one of our younger surgeons to obtain a biopsy sample of one of these nodules, a nodule which the radiologists had called metastatic choriocarcinoma, and villous structures were identified.

These opinions expressed on the subject are based on a personal study of 40 moles and 13 cases of choriocarcinoma plus the wading through of volumes of literature (there are more than 1,000 articles on tumors of the trophoblast). Boiled down to the nonsaponifiable residue this material has given me the following philosophy:

1. True choriocarcinoma is a rare disease. It is occasionally curable. The vast majority of patients so afflicted succumb within 1 year of the time of onset, but delayed metastases may make their appearance as long as 5 years following removal of the primary growth. Resection of solitary or even multiple pulmonary metastases occasionally may avert a fatal outcome.

2. Hydatidiform mole is overtreated. I know of only 2 cases of what we might call metastasizing mole which killed the patient. Regardless of the anaplasia shown by a mole the diagnosis of choriocarcinoma should not be rendered provided any villous structures are present.

3. "Lung shadows," vaginal nodules, and broad ligament extensions in penetrating moles are rarely fatal. They will regress under roentgen-ray treatment, hormonal therapy, or watchful waiting. More of these mysterious benign metastases should be removed for study for a better understanding of the process.

This, in my opinion, is a choriocarcinoma following a hydatidiform mole.

Submitted Diagnoses.—Choriocarcinoma, 36; chorioadenoma destruens, 5; syncytial endometritis, 3; infiltrating mole, 1; benign choriocarcinoma, 1.

Dr. Dockerty: Thirty-six out of 46 diagnosed choriocarcinoma. I would rule against chorioadenoma destruens primarily because of the absence of villi. It can be a fatal disease because it penetrates and can cause fatal hemorrhage or peritonitis, but it is not a metastasizing tumor. I would object to the diagnosis of syncytial endometritis, because every woman who has a baby has some syncytial endometritis. Some of the syncytial cells wander down a little way to the uterine wall; sometimes there is an overproduction of them, and invasion is deep, and infection goes with it. Syncytial endometritis, in my opinion, is not a neoplasm at all. Infiltrating mole to me is synonymous with chorioadenoma destruens. Perhaps the person who made the diagnosis of benign choriocarcinoma knew that the patient was alive 4 years later, and should not have been with a choriocarcinoma. I maintain that you can cure some choriocarcinomas, and I hope that this was one that was cured. It is interesting that it followed the passage of a mole. Fifty per cent of choriocarcinomas do; 25 per cent follow full term delivery; and 25 per cent follow an abortion.

Dr. Manhoff: What are the approximate odds that this woman is now cured or that she may later develop metastases?

Dr. Dockerty: In the literature there are perhaps 10 cases in the entire world of patients who have had delayed metastasis from choriocarcinoma after more than 3 years, so her chances are excellent—99 per cent, perhaps.

Dr. Leo Lowbeer, Tulsa, Okla: Have you any conception or philosophy, in these cases of delayed metastases, as to what may have happened to the tumor in the silent interval of 3 or 10 years?

Dr. Dockerty: I do not. These tumor cells are strictly under the influence of important hormones that we do not know enough about. For instance, cancer of the prostate is under the influence of male hormones, and this is under the influence of pregnancy hormones. I suppose the hormones keep these cells quiet until the patient reaches a period in life in which there is a change in the hormonal balance; then some trigger mechanism is released and the tumor recommences to grow. Of course this is observed in other forms of cancer, too, and I have seen a cancer of the breast sit around in a patient for 35 years, and then kill her with a brain metastasis.

Dr. J. J. Andujar, Fort Worth: I am interested in the diagnosis of chorioadenoma destruens, since it happened to be the horse I was riding, and this diagnosis would be perfectly compatible with what has happened.

Dr. Dockerty: Well, for the sake of argument, let us change the title and call it penetrating mole. In the first place the original mole was not penetrating. In the second place, when the uterus was removed there was no evidence of a mole at all. I have seen 3 or 4 of these cases of chorioadenoma destruens, and the microscopic picture that I have seen was that of red nodules deep in the uterine wall. Most people would have called them choriocarcinoma; and yet the red nodules had villi in them. They are more dangerous than moles, because they cause serious and sometimes fatal hemorrhage and become infected. For chorioadenoma destruens or penetrating mole I require evidence of a mole; whereas in choriocarcinoma, if I find any villi whatsoever, I will not give it a "malignant" diagnosis.

CASE 15

Diagnosis.—Cellular degenerating fibromyoma.

Contributors.—Dr. D. A. Todd and Dr. A. M. Richmond, Nix Hospital Clinical Laboratory, San Antonio.

History.—Pelvic examination of a 45 year old white woman revealed numerous firm nodular masses in the cul-

de-sac, which at surgery were found to be separate from the ovary and tubes, but attached to the posterior vaginal wall and cervix. These masses of firm gray tissue varied in size and shape, the largest measuring 8 by 6 by 2.5 cm. Cut surfaces were mottled, gray, and mucoid.

Dr. Dockerty: The two microscopic features of this tumor consist of a very cellular spindle cell growth in some areas and in others of hyaline thick walled blood vessels coursing through edematous, degenerating, and acellular zones. Even without the aid of special stains it would appear that the spindle cells are smooth muscle elements. Myofibrils are evident, the cytoplasm is somewhat basophilic, and the growth pattern features bands and bundles. In the more cellular zones the nuclei are crowded together and their chromatin content is impressive; yet careful search fails to reveal mitotic figures. An origin from the cervix seems likely.

In the more acellular areas numerous thick walled vessels show hyalinization of their walls. Some thin walled spaces are stuffed with red blood cells, and others show thrombosis with organization and recanalization. In the edematous fibrous tissue about these vessels only a few viable smooth muscle cells are to be found. It may be disturbing to some that peripherally the growth seems to be invading fat.

Although some pathologists might wish to call this tumor hemangiopericytoma, I do not feel that such a diagnosis is tenable. To me the main problem concerns the possibility of malignancy in what is obviously a smooth muscle tumor. Like Dr. Stout, I feel that the work of Evans is extremely helpful in this regard. With mitotic counts of less than 1 in 10 high, dry fields the evidence of clinical recurrence and of metastasis is negligible in cellular fibroids. Even the presence of giant cells means little unless mitoses are present. Moreover, one must be certain not to mistake certain pyknotic nuclei for mitoses in these growths. The mitotic figures to be significant must be genuine. In my experience degenerating fibroids do become cellular, especially the rare cervical variety. They can become frighteningly cellular, but without mitoses my fright does not become chronic.

I certainly do not have the answer as to what induces this cellularity, although I have felt for some time that anoxemia was important in etiology. The female sex hormone estrogen with its periodic change in titer has a profound effect on blood vessels, particularly of the uterus. The effect is best seen in what we call red degeneration. Vasospasm short of producing complete infarction in some strange way seems to induce increased cellularity.

The only other lesion that I have ever seen which remotely resembled this was in the pelvis, and was not a fibroid. It was a benign pelvic fibrous peritoneal mesothelioma, comparable to the ones seen in the thorax. Primary tumors of the peritoneum are usually malignant and papillary with the production of mucoid ascitic fluid. The same lesion in the chest will form fibrous nodules or produce a lesion which resembles a smooth muscle tumor. There are occasional exceptions to this rule. I do not think that mesothelioma is the diagnosis in this case, because of the smooth muscle appearance of the cells, the hyaline changes around these blood vessels, and the fact that there were large areas of degeneration in the tumor. I did not see any clefts; I did not see any papillary formations. Is there any subsequent history on this particular case?

Dr. A. M. Richmond, San Antonio: Seven months after operation the patient was doing very well. Some have called this "leiomyoma peculiare."

Submitted Diagnoses.—Leiomyoma, 18; leiomyosarcoma, 17; malignant Schwannoma, 3; fibroma, 1; neurofibroma, 1; hemangiopericytoma, 1; neurilemoma, 1; sarcoma botryoides, 1; pseudomyxoma peritonii, 1; endometriosis, 1.

Dr. Dockerty: Quite a number were disturbed, apparently, by the cellularity and called it leiomyosarcoma. There is some palisading in this tumor, which would make one wonder about a neurogenic origin; but I do not think it is either a Schwannoma or a neurofibroma. I mentioned before that myomatous tumors of the gut have a tremendous tendency to palisade like a neurofibroma; the uterine ones ordinarily do not. Hemangiopericytoma probably was suggested by the appearance of the vascular zones, and yet we noticed in areas that the vessels were very hyalinized and there were no pericytes, so I think that we can rule out hemangiopericytoma. We have seen a fair number of cases of sarcoma botryoides, and although I have seen them recur and involve the peritoneum, they do not appear as tumors on the posterior portion of the cervix without bleeding. It has always intrigued me how babies get these tumors in their vaginas; little boys get them in their prostates; young women get them in their cervices; and in patients over the age of 60 they are nearly always endometrial tumors. Sarcoma botryoides, moreover, is a highly malignant growth, and this case is at most borderline as regards malignancy. The diagnosis of pseudomyxoma peritonii was probably influenced by the gross description, because certainly we did not see any mucous cells. In males the principal cause for pseudomyxoma peritonii is a malignant mucocele of the appendix. In women the commonest cause is a malignant, and usually a ruptured, mucous cancer of the ovary. I would rule out endometriosis as the stroma does not resemble that of the endometrium and there are no glands.

CASE 16

Diagnosis.—Seroanaplastic carcinoma of ovary.

Contributor.—Dr. J. J. Andujar, Fort Worth Medical Laboratory, Fort Worth.

History.—A 60 year old woman had a progressively enlarging cystic mass in the pelvis, which at surgery appeared to be ovarian in origin. The specimen removed measured 13 cm. in diameter, was nonlobulated, and contained sessile, grayish white projections on its inner surface.

Dr. Dockerty: I should like to discuss this case under two headings: first, the wide spectrum of primary epithelial neoplasms of the ovary, and second, the difficulty of distinguishing primary from secondary ovarian cancer.

This neoplasm is so anaplastic that it almost defies classification. Here and there throughout the growth, however, and especially towards its periphery are noted clefts lined by columnar cells, some of which appear to be practically benign. Transitions between such elements and the solid nests of tumor cells are apparent. The growth, then, is basically glandular, and according to the Broders method of classification is a grade IV adenocarcinoma. In the Barzilai terminology it could be listed as a seroanaplastic carcinoma. The implication is that it began as a serous cystadenoma, but the proof is difficult to obtain.

Some of you may have entertained the thought that since the cells were small and dark staining with very little cytoplasm, the diagnosis of highly malignant granulosa cell tumor was in order. In a study of more than 100 granulosa cell tumors, however, I have been so impressed with the ability of examples showing many mitoses to "function" and produce large uteri with fibroids, that on this basis alone I would be inclined to rule out that diagnosis. The presence of glandular spaces of course is the strongest "histologic" argument for placing the tumor in a category other than that of malignant granulosa cell tumor.

The growth could represent adenocarcinoma in a teratoma, and were the tumor a testicular one, such would be the most acceptable diagnosis. In ovarian tumors of this type, however, multiple sections usually will bring out the tridermal derivation of the growth. Teratocarcinomas of the ovary comprise but a small percentage of ovarian malignancies, and they usually occur among younger women.

I do not believe that mucinous carcinoma of the ovary is a likely diagnosis. In a study of 350 mucinous ovarian tumors at the clinic, Cariker and I found about 130 that were malignant. There were no grade IV lesions in the series, and only 4 of the growths were grade III mucinous adenocarcinomas. By a similar token we can almost rule out endometrial-like carcinoma of the ovary such as is known to complicate endometriosis. Tumors of the type seen in this case are all too frequent, and they account in large part for almost 50 per cent of ovarian cancers. They are so fixed at the time of operation that one has to speculate even with regard to their ovarian origin. The diagnosis of grade IV adenocarcinoma, primary probably in the ovary, is a fairly accurate estimate as to how definitive we can get in classifying such growths.

This brings me to the second point of the discussion, namely, the impossibility of deciding at times when a given ovarian tumor is primary or metastatic. The uterus, the colon, the pancreas, and the stomach are just four of the parent sites which can give rise to unilateral or bilateral, solid or cystic, encapsulated ovarian masses which have the gross configuration of primary ovarian tumors. The ovary acts the part of the perfect host to such an extent in some of these cases that it almost takes intuition to interpret the situation correctly. If on a fresh frozen section at the time of surgery one sees melanoma, lymphosarcoma, or grade IV mucous adenocarcinoma in an ovary, the growth is metastatic. In all other instances except where papillary cancer with psammoma bodies makes fairly certain a primary origin in the ovary, the surgeon should search carefully for the possibility of another "true" primary growth.

In our series of ovarian tumors the grade IV cancer cases were frequent. In table 1 are some of the different types of ovarian epithelial malignant new growths.

TABLE 1.—*Prognosis for Ovarian Carcinomas.*

Tumor Type	Patients	5 Yr. Survival	
		No.	%
Endosalpingoma	45	30	67
Mesonephroma	8	4	50
Mucous cystadenocarcinoma	30	15	50
"Endometrial" type of adenocarcinoma	45	10	22
Teratoblastoma	6	1	17
Seroanaplastic carcinoma	74	12	16
Indeterminate type	20	0	0
Total	228	72	32

What we mean by endosalpingioma is a low grade, papillary, serous cystadenocarcinoma of the type associated with psammoma bodies. The mesonephromas were the rare type described by Schiller. Mucinous cystadenocarcinoma is fairly frequent and has a very good outlook if you can get it when it is encapsulated. Ruptured, this tumor is the one that gives rise to "jelly belly." In the endometrial type of ovarian cancer I found quite a few in which the microscopic appearance was reminiscent of adenoacanthomas of the endometrium. In a study of 45 of these adenoacanthomas of the ovary I found that more than a third of them had what appeared to me to be an independent cancer of the endometrium, as though some carcinogenic influence had simultaneously hit the endometrium that lined the uterus and the endometrium

that occurred in these ectopic foci. The fact that the 5 year survival was good in this group of 40-odd cases of adenoacanthoma indicated to me that the uterine lesion was not indeed metastatic. When cancer of the ovary metastasizes to the endometrium, as it does in 5 per cent of cases, the prognosis is very, very bad. The prognosis is bad because the growth starts multicentrically in endometriosis, in many instances, and disseminates all over the peritoneal cavity. Highly malignant teratomas were diagnosed by making multiple sections and finding different types of tissue —again, a prognosis was bad; but the series is very small. The seroanaplastic carcinomas, usually grade III or IV, have a bad prognosis. The type was difficult to classify in 20, and in this small series there were no 5 year survivals. The over-all survival, including patients treated by surgery and irradiation, is 32 per cent.

Submitted Diagnoses. — Adenocarcinoma of ovary, 16; granulosa cell tumor, 4; malignant granulosa cell tumor, 6; teratocarcinoma, 2; carcinoma of fallopian tube, 6; Brenner tumor, 1; miscellaneous malignant tumors, 6.

Dr. Dockerty: We have already discussed granulosa cell tumors and teratomas. Carcinoma of the fallopian tube is a possibility. Carcinoma of the fallopian tube derives from epithelium that may occur ectopically in the ovary, and ovarian and tubal carcinomas may look much alike, even to the formation of psammoma bodies. We have not actually ruled out the possibility of this growth being primary in the tube; but I will say this, that it is much more common to have ovarian cancer involving the tube and producing a picture of malignant hydrosalpinx than the reverse. In a series of more than 4,000 ovarian and tubal malignancies we have seen only about 24 in which the primary was present in the tube. These cancers, before they become large, metastasize to the ovary; but the tubal component is nearly always considerably larger than the ovarian secondary. As for Brenner tumors, I have not seen or read from the literature about examples that were very anaplastic.

Dr. L. R. Hershberger, San Angelo: We recently have had a case of endometrial carcinoma that at surgery also showed ovarian carcinoma. Is that a blood borne metastasis, or is it an implantation via the tube?

Dr. Dockerty: I once thought that cancer of the ovary ordinarily spread to the endometrium by lymphatic or direct extension, but I believe more and more now that it is by transit through the fallopian tube. There are recent studies in the journal *Cancer* to show that if one washes out the fallopian tubes in cases of uterine cancer (where it is twice as difficult for the cancer cells to spread from the endometrium to the ovary as the reverse), one finds positive smears in more than 50 per cent.

CASE 17

Diagnosis.—Ovarian masculinovoblastoma.

Contributor.—Dr. L. J. Manhoff, Jr., Robert B. Green Hospital, San Antonio.

History.—A 40 year old woman was admitted to the hospital because of abdominal pain. She had increased growth of hair of male distribution which necessitated shaving almost daily, and she had had no menses for 8 years. She had a blood pressure of 160/100, a deep masculine voice, a heavy muscular body, an enlarged clitoris, and a mass in the left side of the pelvis. Urinary 17-ketosteroids were 55 mg. in 24 hours and after administration of ACTH increased to 100 mg. Corticoids and fasting blood sugar were normal. Glucose tolerance curve showed a peak of 248 mg. per 100 cc. in 1 hour and return to normal in 2 hours. At surgery a 520 Gm., lobulated, yellowish tumor was found replacing the left ovary.

Dr. Dockerty: I believe Dr. Manhoff has some gross pictures in this case.

Dr. Manhoff: These Kodachromes are through the courtesy of Dr. J. W. Goldzieher.

The clitoris is considerably hypertrophied and there is marked hirsutism. I believe the patient was shaving every day. The tumor grossly was lobulated and the cut surface showed a variegated appearance. Grossly the ovary was not identifiable, although in some of the sections there is ovarian tissue identifiable in the edge of the tumor.

Dr. Dockerty: In this case we are presented with a large, solid, hemorrhagic, yellow tumor of the ovary. Microscopically one is struck by the composition of large clear and smaller granular cells which have an epithelial appearance and an arrangement in alveolar clusters. There is an intimate relation between the tumor cells and the lumens of the numerous and very thin walled blood vessels. This picture is reminiscent of hypernephroma on the one hand, and certain tumors of the ductless glands on the other.

The cytoplasm of the cells contains lipoid material. The nuclei are round, somewhat hyperchromatic, and possessed of sharply defined nuclear membranes. Nucleoli are large and mitotic figures are not difficult to find, especially in cells which appear more anaplastic than their neighbors.

Without any knowledge of the clinical history I believe that we can rule out such things as hypernephroid carcinoma of the ovary, which to my mind is an ordinary solid cystadenocarcinoma with hydropic cells. These growths are much more active than the tumor under consideration, and they lack the beautiful alveolar arrangement of cells displayed by this case. Metastatic hypernephroma can be excluded for similar reasons. With three remaining choices, namely, luteinized granulosa cell tumor, Leydig cell neoplasm, and adrenal-like carcinoma of the ovary, one turns to the history for assistance. Since generous sampling of the tumor fails to disclose typical areas of granulosa cell tumor we are not surprised that this patient is masculinized. Remaining to be an-

swered is the question of testicular versus adrenal histogenesis.

The literature contains 20 cases of large yellow ovarian tumors associated with Cushing's syndrome, and I personally have studied and reported 1 such case. Leydig cell tumors give pure masculinization. They do not induce osteoporosis, plethoric obesity, hypertension, striae, diabetes, and so forth. Unfortunately in this case the clinical picture is strongly flavored with masculinization. I believe that the hypertension and the hemoglobin reading of 17.3 Gm. point strongly to an effect of hormones other than testosterone. I wonder if the patient's abdominal pains did not include backache, and if roentgenograms of the long bones and of the spine would not have shown osteoporosis? Positive evidence in this regard would be decisive, since testosterone, the product of interstitial cells, puts protein into the bones, producing sclerosis, whereas the adrenal hormones produce osteoporosis. The high blood sugar peak with the prompt return to normal signifies that no diabetogenic hormone was being produced. The ketosteroid levels are very high for an adrenal tumor, and the normal values for corticosteroids somewhat disturbing.

We can say then that in this case the clinical and laboratory picture is mixed, but is predominantly that of virilization. We must decide the issue on the basis of pathology alone. My vote is for adrenal rest carcinoma for the following reasons:

1. The gross tumor is large and yellow. Most of the virilizing testicular rest tumors that I have seen or read about have been small hilar nodules, often bilateral.

2. Microscopically, I think that the cells are larger than Leydig cells. Their alveolar arrangement is identical with that seen in adrenal tumors with Cushing's syndrome.

3. I believe that Teilum and others have gone too far in ascribing a testicular origin to ovarian growths of this type and then blinding themselves to the fact that the tumors produce hormones which are native only to the adrenal.

Submitted Diagnoses.—Masculinovoblastoma (adrenal rest tumor), 36; adrenal cortical carcinoma of ovary, 3; arrhenoblastoma, 3; dysgerminoma, 1.

Dr. Dockerty: It seems that masculinovoblastoma is the choice of the majority, and this should include those who called it an adrenal cortical carcinoma. I have already attempted to rule out an arrhenoblastoma, and I could not buy this for a dysgerminoma, which in my terminology is a growth which microscopically is a dead-ringer for seminoma of the testis. Dysgerminomas consist of very anaplastic cells which one cannot ascribe to an epithelial or to a connective tissue origin; they are large polygonal cells arranged in a mosaic pattern without glands or papillary formation, and interspersed here and there with nests of lymphocytes. We did not see any lymphocytes in this tumor, and the cells were clear instead of dark. Moreover, dysgerminoma is a so-called "neutralizing" tumor, although there are 2 or 3 examples in the literature of masculinizing dysgerminomas. I think, however, that if these cases were analyzed further, adrenal and/or testicular tissue would be found intermingled with the other elements.

Dr. Manhoff: This was Dr. Goldzieher's patient, and as endocrinologist and director of the hormone research laboratory at the Southwest Foundation for Education and Research, he has done extensive hormone studies. I particularly asked him the question whether his studies could help solve the problem as to whether these tumor cells are actually true adrenal cells, as has been long argued in the literature, or whether they are just hormone producing cells that resemble adrenal cells. I think he also has some interesting current follow-up data.

Dr. J. W. Goldzieher, San Antonio: We should have been able to make the correct diagnosis in this case on the basis of the urinary hormone studies. The control level of ketosteroid excretion ranged about 50 mg. a day, and because we are doing some research on hirsutism of various kinds, we ran a number of additional tests, one of which consisted of the injection of 60 units of ACTH gel. To our great surprise the urinary 17-ketosteroids rose to 100 following this injection, which is quite a response. There was also no cortisone suppression, which meant that adrenal hyperplasia was not responsible for this syndrome. I think, with hindsight, these tests indicated that there were partly autonomous adrenal-rest cells which were still capable of responding to ACTH with an increased production of the 17-ketosteroids. I have not combed the literature, but I do not know of any other similar tumor that has been tested in this way. During the postoperative follow-up the hormone levels went down to a normal range; a great deal of the facial hirsutism disappeared; and the patient shaved once a week instead of once a day. Lately, however, her hormone levels have shown a progressive rise, so I think there is either a recurrence or a metastasis. One very interesting thing which is not yet in the literature is that the group at the Worcester Foundation in Massachusetts, as well as the Massachusetts General Hospital group, have been incubating adrenal and testicular tumor tissue with radioactive-carbon-labeled steroids, and to their great surprise they have found that interstitial cell tumors of the testis were able to produce chemical changes, such as hydroxylation at position 11 of the steroid molecule, which are known to be characteristic of the adrenal. It may be that there will be cases in which the histologic appearance is typical of adrenal or interstitial cell, and the chemistry will be

quite the reverse; so at the moment there is a great deal of interest in what these tissues possess in terms of hydroxylating enzymes.

Dr. Dockerty: This particular growth is very unusual, and when I reported our case with Kepler a few years ago there were about 18 in the literature. I do not know how many have been reported since the advent of the steroids, but I think Dr. Goldzieher's studies are probably the first of their kind. I certainly hope that the chemists and the hormonologists will straighten us out on some of these tumors, because interstitial cells of the testis can look a good deal like cells of the adrenal cortex. Maybe histopathology is not the answer in some of these cases.

Dr. Leo Lowbeer, Tulsa, Okla.: How many of these tumors behave malignantly?

Dr. Dockerty: You cannot answer that from the literature, because a surgeon usually reports these cases, frequently before the patient gets out of the hospital, and there is no follow-up report. All that I can say is that our "series" of 1 case was not associated with metastasis, but microscopically it was malignant, and I would not be surprised if there were a recurrence.

Lt. Col. J. H. Hansen, Brooke Army Hospital: In Dr. Iverson's series reported in 1947, 2 out of 17 cases had metastatic lesions.

Dr. Dockerty: That is interesting, but I am sure that we do not have adequate follow-up data on the majority of cases, for the reason that they are reported too early.

CASE 18

Diagnosis.—Malignant Brenner tumor.

Contributor.—Dr. Dockerty.

History.—A 59 year old white woman complained of abdominal enlargement of 2 weeks' duration. Sixteen years earlier a 4 cm. nodule found on pelvic examination had been regarded as an asymptomatic fibroid. At operation a fist sized, partly cystic and partly solid left ovarian tumor was removed along with uterus, tubes, and the remaining ovary.

Dr. Dockerty: This neoplasm had a relatively small solid component, attached to which was a bilocular cystic mass. At the junction of the solid and cystic areas the lining had a shaggy appearance suggesting a papillary cystadenoma or cystadenocarcinoma. Every once in a while a fibroma will be seen in the wall of a cystadenoma, and that is what was expected in this case.

The solid portions of this tumor were very firm and fibrous. Microscopically they contained the usual islands of pale epithelial cells with grooved nuclei which we have all come to recognize as being typical of Brenner tumor. Other tissue sections taken from the papillary areas which comprised the soft marshy backwaters of the large central cyst, however, showed a picture reminiscent of papillary transitional cell carcinoma of the urinary bladder. The stroma, so dense in the solid portions of the tumor, was here reduced to delicate frills which form tenuous supports for many layers of epithelial cells. The latter, while retaining their pale color, polygonal outlines, and grooved nuclei, are distinctly more anaplastic than their "kissing cousins." Mitotic figures can be found to the tune of one in every three or four fields. There is cellular pleomorphism. My diagnosis is malignant Brenner tumor.

With lenient judgment we perhaps can accept 6 cases of malignant Brenner tumor among the hundreds of Brenner tumors reported in the literature. The 2 patients reported by Von Numers died of their malignancy, but without the benefit of necropsy. Rawson's patient exhibited extensive local spread with involvement of the right kidney and ureter. The fate of 3 other patients is unknown. Our case is a recent one in which the malignancy was completely removed, and data on Colvin's case (unpublished) are not available as to outcome.

It would appear, then, that the epithelium of the lowly Brenner tumor can assume aggressive growth characteristics and produce metastasis. Soft succulent areas or papillary features characterize the aggressive variants. The change can affect solid Brenner tumors as well as the type associated with mucinous cysts.

Submitted Diagnoses.—Dermoid cyst of ovary, 5; malignant Brenner tumor, 3; Brenner tumor, 11; teratocarcinoma, 4; functioning feminizing tumors, 5; functioning masculinizing tumor, 1; adenoacanthoma, 4; cystadenocarcinoma, 2; undifferentiated carcinoma, 5; transitional cell carcinoma, 1; Walthard nest cyst, 1.

Dr. Dockerty: Apparently those who diagnosed dermoid cyst were impressed by the squamous epithelial characteristics of this tumor. It has been observed that in Brenner tumors the epithelium is squamous-like, but it lacks epithelial bridges and keratohyaline granules, resembling transitional epithelium more than squamous epithelium. A small minority appears to agree with my diagnosis; and several were not impressed with, or did not commit themselves as to, malignancy. Teratocarcinoma was no doubt diagnosed because of the mixture of benign and malignant elements. As a matter of fact, it has been suggested that the Brenner tumor is part of a teratoma, inasmuch as one-fourth to one-third of the cases are found in the walls of mucinous cysts of the ovary. As for a functioning feminizing tumor, we must recall that Brenner tumor in the past has been confused, and still is confused, with granulosa cell tumor of the ovary. The malignant portion of this tumor was cellular like a granulosa cell tumor, but the benign portion was typical of a Brenner tumor. I would find difficulty in reading functioning masculinizing tumor into this lesion, and I saw nothing resembling

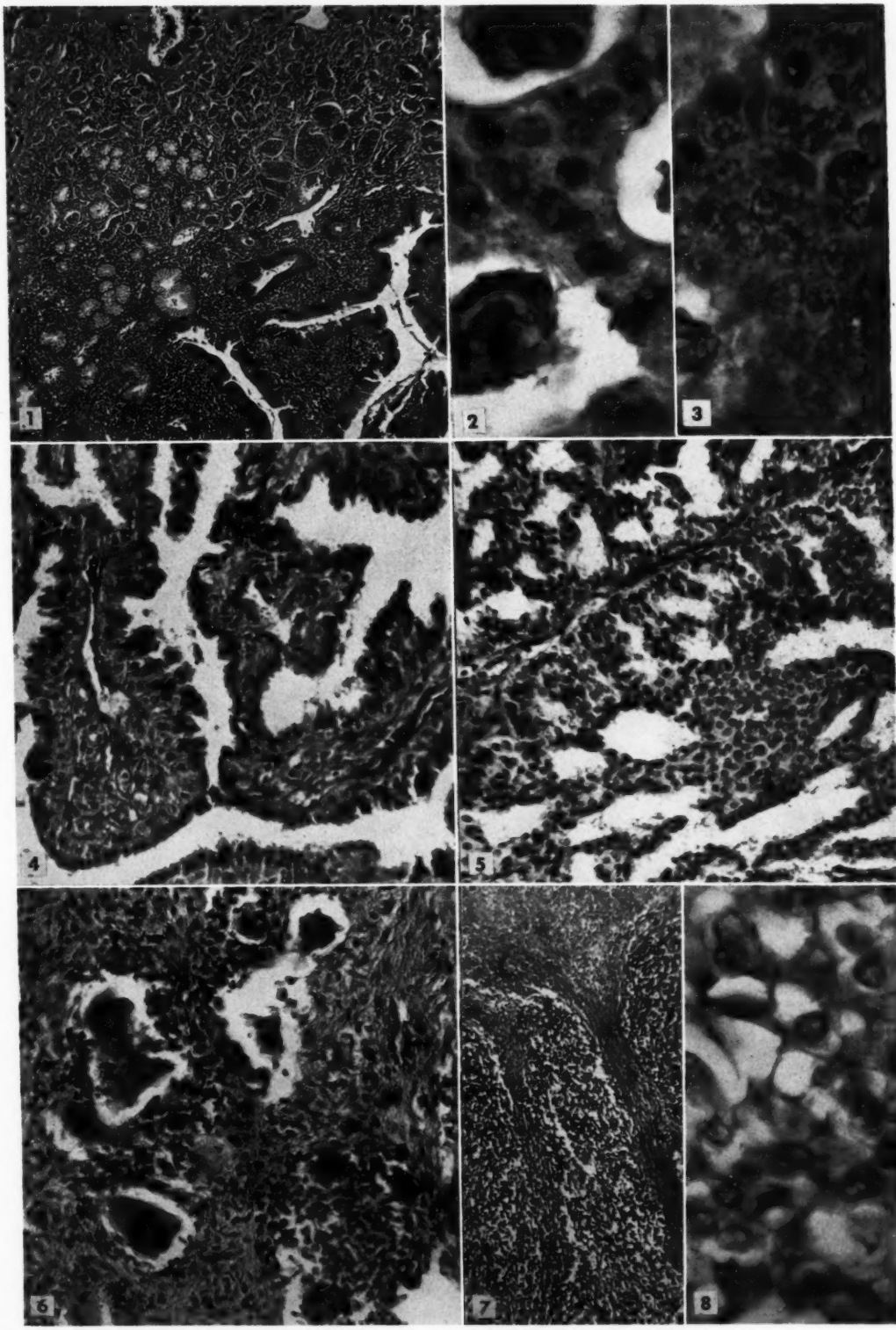


Fig. 1. Case 1. Carcinoid of duodenum. x 60.

Fig. 2. Case 1. **Fig. 3.** Case 19. Carcinoid in dermoid cyst of ovary with Biorck-Thorson syndrome. Both are composed of uniform prismatic cells with pink cytoplasmic granules. x 760.

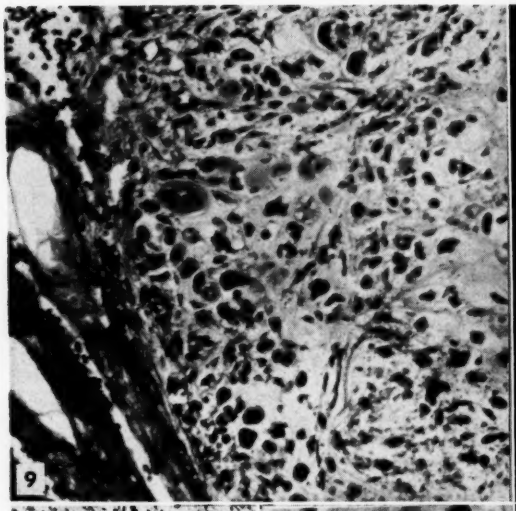
Fig. 4. Case 2. Benign papillomatosis of breast. The papillary stalks have connective tissue cores, and the epithelial elements are uniform with little stratification.

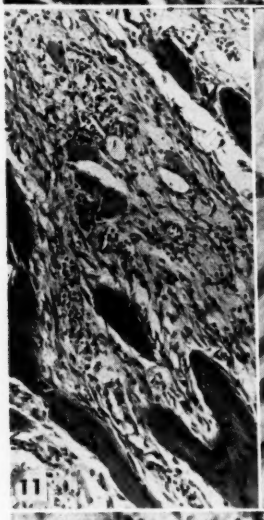
Fig. 5. Case 3. Papillary carcinoma of the breast. As compared to Case 2, the growth is more solidly epithelial and the connective tissue stalks more tenuous. x 170.

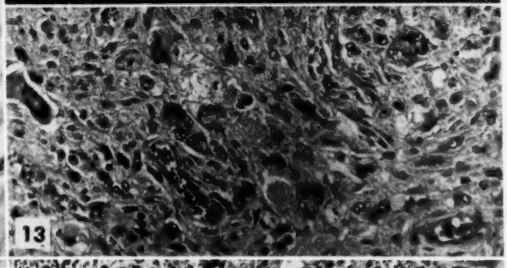
Fig. 6: Case 4. Benign giant cell tumor of scapula. Giant cells are numerous and contain large numbers of nuclei. x 170.

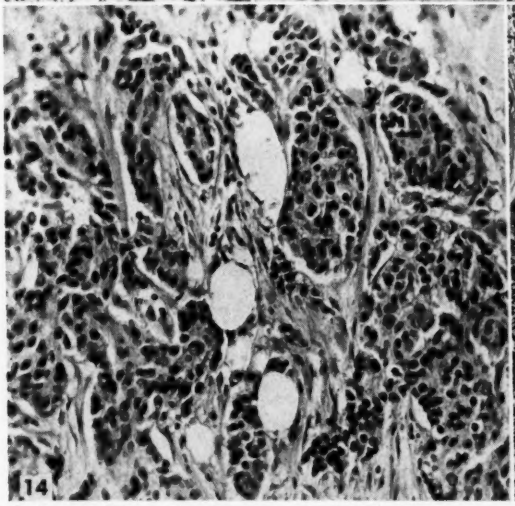
Fig. 7. Case 5. Grade IV chondrosarcoma of scapula. The lesion is cellular with areas of necrosis. x 60.

Fig. 8. Case 5. More or less rounded cells with many mitoses and a surrounding clear zone. x 760.









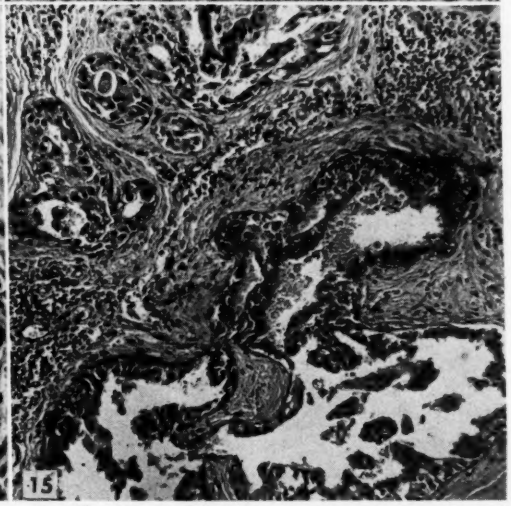


Fig. 9. Case 6. Primary chondrosarcoma (grade I-II) of humerus. The lesion is cellular with plump nuclei, and the matrix is chondromucin. x 170.

Fig. 10. Case 7. Parosteal osteogenic sarcoma of the humerus. Cross section of upper humerus showing the ensheathing tumor.

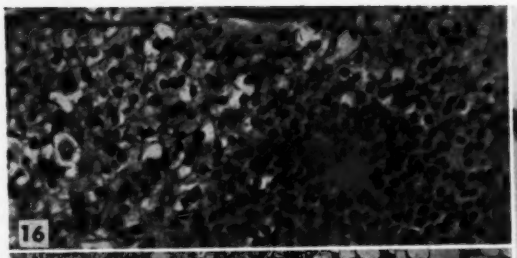
Fig. 11. Case 7. Immature bone in the form of long narrow trabeculae with spindle cells tending to run parallel to them. x 105.

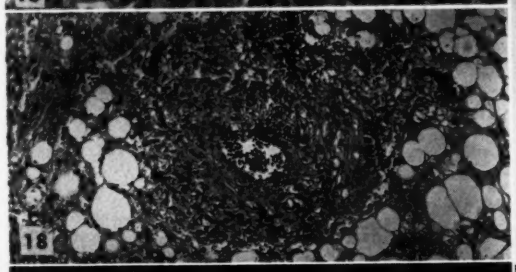
Fig. 12. Case 7. Fibroblasts showing moderate pleomorphism and mitosis (center). x 475.

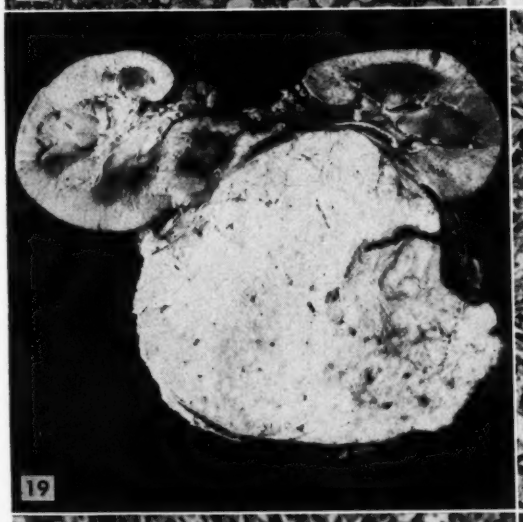
Fig. 13. Case 8. Bilateral metachronous mammary carcinoma and associated metastasizing liposarcoma. This figure shows the anaplastic liposarcoma with giant and bizarre cells. x 105.

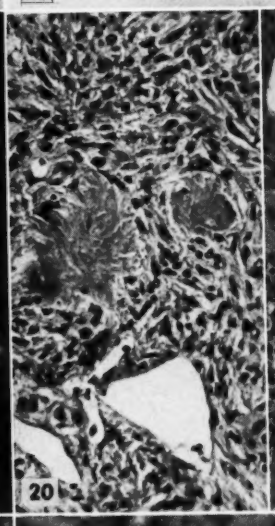
Fig. 14. Case 8. Grade IV scirrhous carcinoma of left breast. x 105.

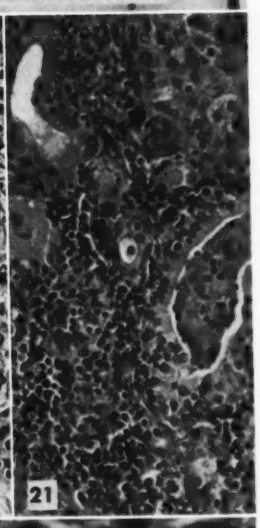
Fig. 15. Case 8. Grade III comedocarcinoma of right breast apparently still in situ. x 170.

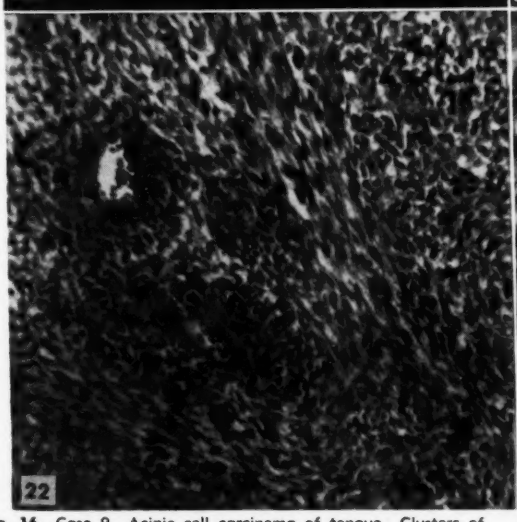












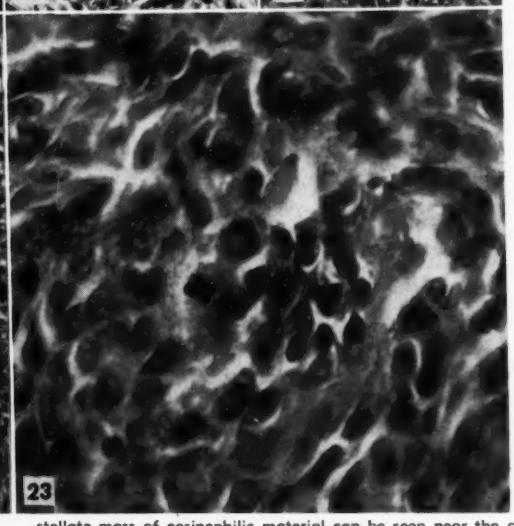


Fig. 16. Case 9. Acinic cell carcinoma of tongue. Clusters of clear cells merge with cells having a granular cytoplasm. x 170.

Fig. 17. Case 10. Multiple angiolipoleiomyomas of kidney. In addition to the large encapsulated tumor mass, arrows indicate the smaller lesions.

Fig. 18. Case 10. Thick walled blood vessels and adipose tissue. x 105.

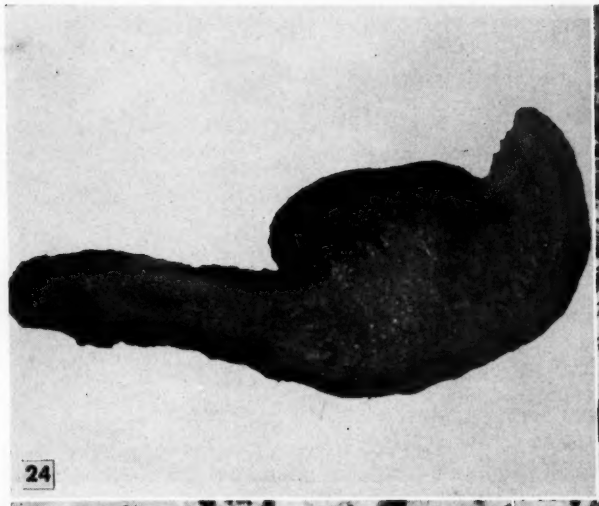
Fig. 19. Case 11. Hypoglycemic tumor of kidney.

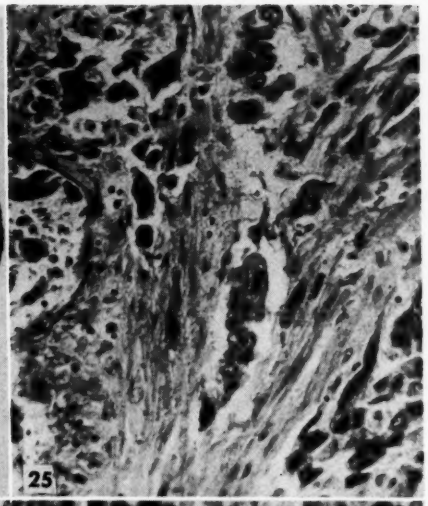
Fig. 20. Case 11. A ball shaped mass and a nearby irregular stellate mass of eosinophilic material can be seen near the center. x 170.

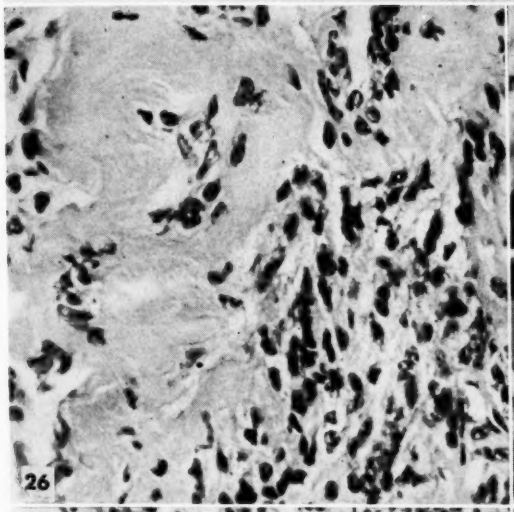
Fig. 21. Case 12. Chronic thyroiditis, mixed type. The intact and disrupted follicles are composed of Hurthle cells. Colloid can be seen in some follicles. x 170.

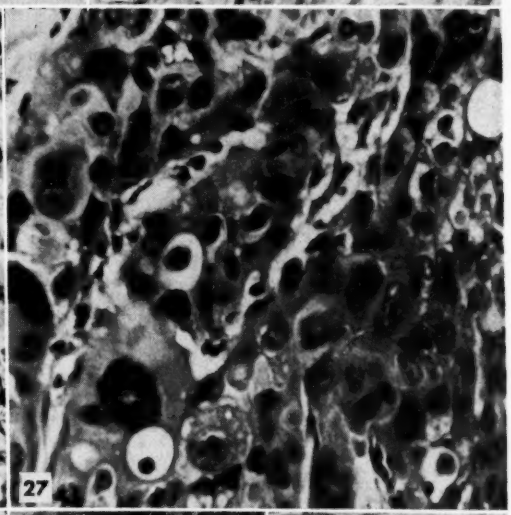
Fig. 22. Case 13. Grade II leiomyosarcoma of the lung. This shows the cellularity and decussating fasicular pattern. x 170.

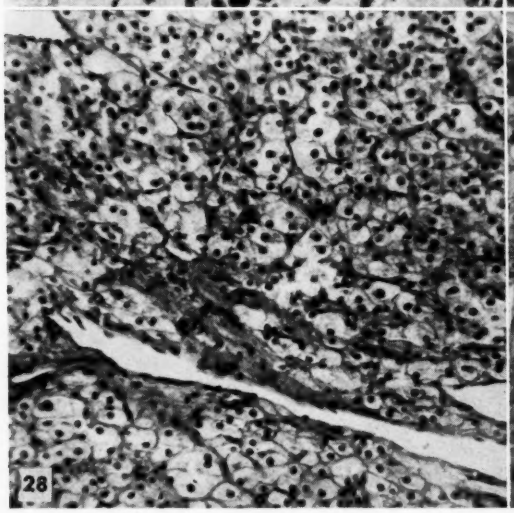
Fig. 23. Case 13. The cells are plump and a mitotic figure is visible. x 760.











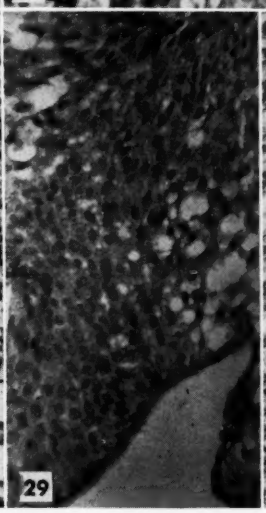


Fig. 24. Case 14. Choriocarcinoma of the uterus. Cross section of uterus.

Fig. 25. Case 14. Anaplastic trophoblasts aggressively invade the uterine musculature. x 170.

Fig. 26. Case 15. Cellular degenerating fibromyoma. Blood vessels with thick, hyalinized walls and cellular smooth muscle elements. x 300.

Fig. 27. Case 16. Seroanaplastic carcinoma of ovary. Marked anaplasia is present in some areas. x 200.

Fig. 28. Case 17. Ovarian masculinovoblastoma. Large clear and smaller granular cells comprise the lesion. x 170.

Fig. 29. Case 18. Malignant Brenner tumor. Area reminiscent of transitional carcinoma of bladder. x 170.

Fig. 30. Case 18. Cellular pleomorphism is present, as well as a mitosis (center). x 475.

interstitial cells. For adenoacanthoma I like to see the glandular epithelium and the squamous epithelium side by side. As a matter of fact, Brenner tumors and adenoacanthomas resemble each other in this respect. In a Brenner tumor the squamous epithelium surrounds the glandular epithelium; in the adenoacanthoma it is the reverse; you get the squamous cells in the center and the glands on the outside. There are some interesting cases wrongly reported in the literature as Brenner tumors, which are typical adenoacanthomas of the ovary. As for cystadenocarcinoma, the lesion was cystic, and there were glands in it. I think, however, the cancer is more like a transitional cell carcinoma than an adenocarcinoma. As for the rest of the diagnoses, I like transitional cell carcinoma best because the malignant cells certainly look like the papillary growths that one sees in the bladder.

CASE 19

Diagnosis.—Carcinoid tumor in dermoid cyst with Biörck-Thorson syndrome.

Contributor.—Dr. Dockerty.

History.—A 50 year old woman had a history of peculiar hot flashes for 2 years. Thirteen years before, artificial menopause had been induced with radium. The hot flashes lasted about 5 minutes following bowel movements and were accompanied by palpitation. During the attacks her skin, particularly over the thorax and face, would become fiery red. For 7 months she had had diarrhea, with 15 or more bowel movements daily. Blood pressure was 235/95. Rectal examination revealed a solid, 10 cm. mass in the cul-de-sac. An attack was precipitated by pressure on the mass.

Dr. Dockerty: We began this seminar with a carcinoid, and it is perhaps appropriate to end it with another. A diagnosis of carcinoid syndrome was made in this case, but because of the size of the mass it was believed that it was a metastasis from a carcinoid elsewhere. A laparotomy was performed with removal of the left ovary, which was the seat of a well encapsulated, somewhat lobulated 6 by 8 cm. tumor which on cut section showed a slight cystic component, but 85 to 90 per cent was solid, lobulated, and yellow. It was palpation of this tumor which induced the attacks of cyanosis, along with the diarrhea.

Microscopically this solid yellow portion of the tumor did not differ materially from case 1, being composed of clusters of uniform prismatic cells with granular cytoplasm. With special silver stains argentaffin granules can be demonstrated in the cytoplasm. The solid yellow portion of the tumor showed the same picture throughout; elsewhere it showed the typical features of a dermoid cyst.

For the first half century of the known history of carcinoids, or "little carcinomas," they were regarded as mere playthings for the pathologist. Next certain obstructive features were recognized clinically, and Miller and Hermann in the early forties described a roentgen-ray sign consisting of acute buckling or angulation of the ileum associated with the presence of a small tumor occurring at the apex of the "kink." Carcinoid was the only tumor which could produce such a phenomenon, but Miller and Hermann were the only investigators who were able to use the sign to advantage, and in 1 case only, at that. An occasionally associated diarrhea was attributed to the effects of obstruction. Patients with concomitant skin changes were sent to the dermatologist or to the allergist, who always came up with diagnoses that confused the issue.

Finally in 1954 Thorson, Biörck, Björkman, and Waldenström tied the loose ends into a syndrome with which you all are familiar and which appears to be more and more common as we review our older series of cases of carcinoid tumors, especially those associated with metastasis.

The hormone produced is apparently serotonin, elaboration of which by the Kultschitzky cells promotes, among other things, normal intestinal peristalsis. Its end product of excretion in the urine is 5-hydroxyindoleacetic acid, or 5-HIAA. It was originally thought that the liver in some manner detoxified the excessive amounts of serotonin produced by carcinoid tumors so that early cases of the carcinoid syndrome were all associated with bulky liver secondaries. These were supposed to have interfered with detoxification. However, Scheifley and I with others have reported instances of the carcinoid syndrome producing death from right-sided heart failure, in which no liver metastasis was present. The present case is a beautiful one to illustrate the point that all one requires for production of the carcinoid syndrome is a large mass of carcinoid tumor tissue, located anywhere in the body. Nonmetastasizing carcinoids do not elicit the syndrome because they are too small. One should always hesitate to designate any tumor as a carcinoid if the size of the primary growth exceeds 1 inch. There are few exceptions to this rule, and this case is one of them.

The present case, so far as I am aware, is the fifth instance of carcinoid tumor in a dermoid cyst of the ovary. Rupert A. Willis reported the first 2 instances of the combination in the early 1940's. I believe, however, that this is the only example of the carcinoid syndrome associated with carcinoid overgrowth in an ovarian dermoid.

Submitted Diagnoses.—Carcinoid tumor, 29; pheochromocytoma, 7; chemodectoma, 3; granulosa cell tumor, ovary, 3; adrenal tumor of ovary, 1.

Dr. Dockerty: Carcinoid tumor appears to be the choice of the majority. Pheochromocytoma and chemodectoma are about the same, and the ones who made these diagnoses were probably thrown off by the granularity of these cells. Cells in a pheochro-

mocytoma are much larger and more pleomorphic than they are in a carcinoid. Possibly both of these tumors are related, though, to sympathetic nerve tissue. Granulosa cell tumor of the ovary is sometimes difficult to distinguish from a carcinoid. Possibly special stains would help, and of course a test of the urine for 5-hydroxyindoleacetic acid would settle the issue. As for adrenal tumor of the ovary, I think perhaps that the cells are too dark and granular for that particular diagnosis.

Dr. F. P. Bornstein, El Paso: Would you know whether this tumor was associated in the teratoma with a mucous membrane resembling either intestinal or respiratory epithelium? I wonder about the histogenesis and whether some of these carcinoids in dermoid cysts are not much more similar to bronchial adenomas than they are to carcinoids.

Dr. Dockerty: In 1 of our first 2 cases we found the gastrointestinal epithelium from which the carcinoid arose; in the other case we did not, but in this particular case the tumor was so very, very large that it was difficult to ascribe the origin to any one point. It certainly was a carcinoid chemically and histologically. I would like to mention that I am not certain that carcinoids of the bronchus are carcinoid tumors at all, and I hope someone will prove it by examining the urine for 5-hydroxyindoleacetic acid.

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Genetic Factors Pertaining to The Etiology of Cerebral Palsy

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THE ETIOLOGY of the majority of the cerebral palsies is well established; however, some forms of this condition are not fully understood, and much research is still necessary to prove or disprove some of the theories on etiology. The science of human genetics offers a new and promising approach to this problem, and it is my purpose in this article to reexamine, elucidate, and possibly amplify the genetic factors pertaining to the etiology of cerebral palsy, since these, although frequently alluded to by various authors[1, 2, 6, 7, 9, 10, 15-17, 19-21] until recently have been the most neglected.

In this discussion the term cerebral palsy is defined as "any abnormal alteration of movement or motor function arising from defect, injury, or disease of the nervous tissue contained in the cranial cavity."[5] Perlstein[17] listed the factors which may precipitate damage to the brain as follows: (1) those which occur in the prenatal period, from the time of conception to the onset of labor; (2) those which

operate in the natal infant; and (3) those which operate in the postnatal period, from the time that the child is viable, throughout the rest of its life. Genes actually exert their influence on a person at all the stages of life referred to above, and it is not inconceivable that cerebral palsy may be due to different gene transmission patterns. Only the sex-linked type of inheritance has been demonstrated, but there also may be recessive and dominant modes of inheritance with varying degrees of penetrance or expressivity that affect the individual's phenotype.

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Research is still needed on the etiology of the cerebral palsies. Human genetics offers a promising approach. Genetic processes, their possible relationship to disease, and the importance of recording in case histories the number and sex of normal as well as affected siblings, both living and dead, for research purposes are discussed.

Minear,[12] in a special article "A Classification of Cerebral Palsy" based on the majority opinion from questionnaires sent to the members of the American Academy for Cerebral Palsy, indicated the following as bases of classification for cerebral palsy numbered according to the Academy's order of preference: (1) motor symptoms, (2) topographic involvement, (3) etiology, (4) anatomical site, (5) severity of involvement, (6) degree of muscle tone, and (7) supplemental data. For purposes of reference, clarity, and convenience I am quoting the points outlined in the third choice of this classification below:

Etiological

A. Prenatal

1. Hereditary—Genetically transmitted and may involve racial or familial predilections and often sex-linked. These are often classified as "cerebral agenesis." The symptoms are often present at birth and generally do not progress. Examples: Hereditary athetosis, familial tremor, familial spastic paraplegia.
2. Acquired *in utero*
   a. Prenatal infection (toxoplasmosis) rubella, or other maternal infection.
   b. Prenatal anoxia—carbon monoxide, or strangulation of mother, maternal anemia, hypotension, e.g., following spinal anesthesia, placental infarcts, or placenta abruptio, kinking, knots or prolapse of the cord.
   c. Prenatal cerebral hemorrhage—maternal toxemia, direct trauma, maternal bleeding diathesis.
   d. Rh factor, Kernicterus due to Rh factor (controversial).
   e. Metabolic disturbances, diabetes.
   f. Gonadal irradiation, harmful exposure to x-ray.
   g. Maternal malnutrition.

Although the motor classification is given preference (as treatment of cerebral palsy is mainly symptomatic), a knowledge of the etiology not only gives the clinician a better understanding of the future treatment and pathogenesis of this condition, but also on the basis of a hereditary history preventive measures may be instituted, at least in the form of eugenic advice to parents with cerebral palsied children. This can be given only after careful observation and analysis of sibships, kindreds, and sporadic cases.

REVIEW OF GENETICS

Before pursuing the hereditary aspects of cerebral palsy and the practical application of human genetics by the medical practitioner in general, a brief, preliminary review, for purposes of orientation, of the fundamental nature of the genetic material is in order.

Modern genetics is based on the gene theory. The *genes*, composed of desoxyribonucleoproteins, are the unit determiners of inheritance and are arranged in linear fashion in the *chromosomes*. Genes and chromosomes possess the property of self-reproduction, and the biochemical activity of the cell is dependent upon this property. A gene may undergo a *mutation* (permanent, heritable change in the gene) and produce daughter genes possessing the mutant configuration. Genes act in their particular environment; thus the substrate and conditions with which and under which they act may cause the same gene or group of genes to give different products. It is also well to keep in mind that not one single gene, but rather many genes working together give rise to certain characters or traits. Conversely many individual genes through complex reactions may effect more than one trait or character (polyphenic or pleiotrophic action of the gene). Another interesting characteristic of genes is that they can manifest themselves at any stage in the individual's development and in different degrees of expression. Some genes interfere with prenatal development and may even exert a lethal effect; others manifest their clinical effect after birth, and some are not manifest until adolescence or even maturity.

Man has 48 chromosomes (recent cytological evidence points to 46[18]), 23 like pairs (the autosomes) and one unlike pair (the sex chromosomes X and Y in the male and XX in the female).

During the maturation of the germ cells the chromosome number is reduced to half, but restoration of the full complement takes place during fertilization, keeping the number of the species constant. Thus a person receives one-half of his potential char-

acteristics from each of his parents. Human beings are a heterogeneous group; no two individuals are alike. However, individuals are homozygous for some traits and heterozygous for others.

In human heredity many disease entities are transmitted by different patterns of inheritance. I shall mention only the more common types of inheritance, since a complete discussion of the different modes of inheritance is beyond the scope of this assignment. The most easily detected hereditary pathological disease entities exist because of autosomal dominant genes. Here the condition manifests itself when the gene is present only once, in the so-called heterozygous condition. The next group of anomalous traits exists as a result of autosomal recessive genes. Here the individual has to be homozygous (gene present twice) before the condition manifests itself, and both of his parents have to be heterozygous for the trait, or carriers. Carriers are persons who possess the potentialities of transmitting a disease which they themselves do not show. The last category to be mentioned is the sex-linked inherited diseases. Here, if the trait is recessive, more males will be affected, as sex-linked genes are located on the X chromosome and have no counterpart in the Y. In the female in sex-linked recessive inheritance the gene would have to be present in both X chromosomes before the condition became apparent. Thus sex-linked recessive inheritance may be transmitted by a phenotypically normal, but genetically "carrier" female to approximately one-half of her offspring, and probably that proportion of sons will be affected. This type of inheritance was demonstrated recently for cerebral palsy.[4]

HEREDITY AND DISEASE

Murphy[14] commented on the paucity of knowledge "concerning the relative frequency of congenital defects in families already possessing one malformed member." This lack of knowledge, he stated, was brought to his attention by the experience of a colleague who delivered a patient of 2 very abnormal children in successive pregnancies, having informed her after the birth of the first that she was no more likely to give birth to a second one than was any woman who had never had such an experience. It must be kept in mind, however, that actually once a family possesses an afflicted member the occurrence of subsequent affected siblings is much more likely. In his excellent investigation on the genetic basis of congenital malformations Murphy[14] drew the following conclusions: "The observations which have been made during the course of the present investigation lead to the general conclusion that gross, human congenital malformations arise solely from influences which affect the germ cells prior to fertilization. No evidence is available to indicate that they result from factors which operate for the first time after fertilization has taken place." However, there is an interplay of genes and environment, and together they influence the expression of so-called anomalies in living organisms. Therefore, even though certain adverse conditions are operating in early development, only certain genotypes will be affected. In this connection something must be said about prenatal factors acquired in utero listed under number 2 in the classification under discussion. Benda[3] devoted part 2 of his book "Developmental Disorders of Mentation and Cerebral Palsies" to such prenatal factors in cerebral palsy as (1) birth injuries and anoxia, (2) infectious diseases, and (3) kernicterus. I shall comment briefly on only two other prenatal etiological factors of cerebral palsy listed as acquired in utero: metabolic disturbances such as diabetes and gonadal irradiation such as harmful exposure to roentgen ray.

It is well known that noxious agents acting during early intra-uterine development may lead to abnormalities indistinguishable from those with a genetic basis. Clinically this may lead to the so-called phenocopy, an environmentally induced disease similar to a genetic disease.

With an increasing proportion of persons with diabetes in the population a great deal of emphasis has been placed on this metabolic error. Obstetricians in particular are cognizant of this condition in regard to complications of pregnancy. Jones[11] in a report covering a 25 year period from 1927 to 1951, comprising 100,962 deliveries, of which 184 were diabetic pregnancies, showed statistically "that congenital anomalies were three times, breech and other malpositions three and one-half times, and prematurity (both living and stillborn) four times the frequency of the last five year rate for the hospital." In some of the studies on various clinical syndromes now in progress in our department the number of siblings, ascendants, and collaterals in the pedigrees suffering from diabetes is very pronounced and disturbing and we believe cannot be overlooked as a possible contributing factor. With the advent of insulin and modern prophylactic and therapeutic medicine to ameliorate this metabolic error it is difficult to estimate or even speculate on the incidence of diabetes mellitus in the general population.

Experimental teratology has become a well recognized science. Along this line on an experimental basis Duraiswami[8] has produced malformations by injecting developing chick embryos with crystalline insulin. It is not difficult to speculate on the effect of insulin shock during early pregnancy. Certainly, dosage and the threshold tolerance of the individual in all such treatments must be of primary consideration.

Ever since Muller's[13] classical discovery in 1927

that roentgen rays produce mutations in Drosophila melanogaster, much attention has been directed to the study of this action. More recently radium, ultraviolet light, and chemical mutagens also have been employed to induce genetic changes. In our present atomic age we are all concerned with the effect of ionizing radiation on the transmission of genetic material. However, gonadal irradiation, harmful exposure to roentgen ray, listed in the etiological classification under prenatal development in utero requires amplification.

Prenatal by definition means the period from the time of conception to the onset of labor. Therefore, gonadal irradiation of the mother for that particular pregnancy would have to be eliminated, as this would have had to occur prior to fertilization in order to produce a defective germ cell. But any alteration of the genetic material in the gonads during pregnancy could affect the progeny of future pregnancies. In addition the gonads of the developing fetus might be damaged by abdominal irradiation of the mother. This type of change, however, would manifest itself in the progeny of later generations. It must be remembered that irradiation could also affect the fetus per se, since the immediate effect of ionizing radiations is an inhibition of mitosis (cell division). Then the cells apparently recover from this insult and there is a burst of mitotic activity. In conjunction with delayed and then accelerated mitosis there is an abnormal distribution of genetic material to the daughter cells. If these damaged cells are early embryonic cells that continue to divide at a critical stage in the developing individual, they may produce an abnormal body region. The extent of damage will depend upon how large a portion of body the affected parent cell produced. If the abnormality of the affected cell is very severe, it may result in the failure to reproduce and/or in its death. It is also possible that gonadal irradiation during pregnancy may inhibit the normal hormone production, thus altering the physiological activity of the mother, and in turn having an effect on the developing fetus.

COMMENT

In reviewing some of the factors in the etiology of cerebral palsy I have endeavored to point the way to a greater understanding of the genetic processes as an application of genetics to the realm of cerebral palsy. It should be reemphasized that the solution to the problem of cerebral palsy requires complete cooperation among the various disciplines. This is especially true in recording case histories. In order for these histories to be of value for genetic analysis they should include the number and sex of the normal as well as the affected siblings, both living and dead. Most records of any inherited condition are virtually useless for genetic analysis because of this lack of information. With such a large number of cases of cerebral palsy of unknown etiology the genetic aspect of this challenging problem can no longer be neglected. Thus, with more complete case histories and data available for statistical analysis, the geneticist as a member of the team attempting to solve some of the problems concerning cerebral palsied children must play a vital part.

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Progress Toward Cancer Vaccine

Evidence has been uncovered at the Veterans Administration Hospital in McKinney which could point the way toward development of a vaccine for treatment of some types of cancer, according to officials. Their work, they say, indicates the existence of a natural defense mechanism of the body against cancer. The researchers now are looking for a way to activate this reaction to the extent it would be useful in destroying some types of malignant growth.

Observations on Sulocarbilate as An Orally Active Carbonic Anhydrase Inhibitor Diuretic Agent

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**With the Technical Assistance of
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SINCE the inception of oral diuretic therapy approximately 5 years ago, the carbonic anhydrase inhibitors have received considerable publicity and have been used widely. The continued research with carbonic anhydrase inhibitors evolves from the limited potency, untoward reactions, and the readily developing tolerance to the agents of this type presently available. This report consists of clinical and laboratory observations on sulocarbilate (p-sulfamyl-2-hydroxyethyl-carbanilate) (W-1548-1), a carbonic anhydrase inhibitor of the sulfonamide type* (fig. 1).

**The product used in this study was designated by the manufacturer, Warner-Chilcott Laboratories, as W-1548-1.*

Dr. J. B. Rochelle and his coauthors submit this paper from the Departments of Medicine and Pharmacology, Baylor University College of Medicine and the Veterans Administration Hospital.

Studies on sulocarbilate (p-sulfamyl-2-hydroxyethyl-carbanilate), a carbonic anhydrase inhibitor, indicate that there is no significant difference in its ability and that of acetazoleamide to increase water and sodium excretion. The optimum diuretic dose is approximately 2,000 mg. daily. No toxicity was observed.

METHODS AND MATERIALS

The technique of this study has been described previously. Briefly, 10 male patients with a roughly equivalent degree of controlled congestive heart failure were the subjects of the study of this drug. These patients were maintained on a metabolic ward under the observation of trained personnel. With the exception of diuretic agents, all medications required for control of their heart failure were continued throughout the study. The patients were allowed to

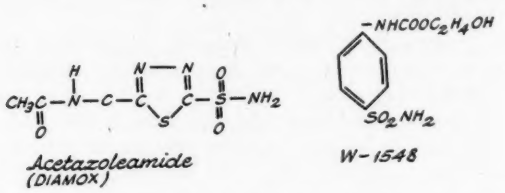

Fig. 1. The structural formulas of acetazoleamide (Diamox) and sulocarbilate (W-1548-1).

be ambulatory about the ward. The sodium content of each patient's diet was exactly 50 milliequivalents per 24 hours. The patients drank 3,000 ml. of distilled water daily. They were allowed to equilibrate on this diet until each patient excreted a constant amount of sodium which approximated 90 per cent (45 meq/24 hours) of their dietary sodium. The diuretic agent to be tested was then administered at 6 a. m., the beginning of the 24 hour period. Before the administration of the next dose of the diuretic agent, several days (3 to 4) were allowed for the patient to regain his body weight by the restoration of sodium and water stores. Thus, the interval between successive drug doses was approximately 4 to 6 days. Each patient was weighed before breakfast and after voiding. Urine was collected in 24 hour periods and analyzed for sodium, potassium, chloride, and creatinine (as a test of the accuracy of the collection). Other routine studies included plasma electrolyte determinations, electrocardiograms, and chest roentgenograms on each patient.

Dosage response relationships were determined on this drug. It was given orally as a single dose starting with 250 mg. and progressively increased (250, 500, 1,000, 2,000, 4,000, 8,000 mg.). No toxicity was encountered in the assay. Since doses of 2,000 mg. effected optimum natriuresis, the bioassay of this group of 10 patients was carried out at dosages of 2,000 and 4,000 mg. as a single oral dose and at dosages of 1,000 mg. twice daily.

A complete electrolyte excretion pattern was obtained on one of the patients. The patient was given 2,000 mg. orally twice at a 12 hour interval (6 a. m. and 6 p. m.). The patient drank 3,000 ml. of distilled water which was allotted fractionally to assure a constant water intake throughout the day. Urine

was collected under oil in six periods per 24 hours (three 2 hour periods and three 6 hour periods). The specimens were analyzed for sodium, potassium, chloride, phosphate, titratable acidity, ammonia, bicarbonate, total solutes, and volume.

RESULTS

The dosage response curve is illustrated in figure 2. One thousand mg. as a single oral dose is not a threshold dose; 2,000 mg. of the drug as a single oral dose is at the apex of the curve for natriuretic response. Increasing the single oral dose in excess of 2,000 mg. effects no further augmentation of natriuresis. This dosage response curve is similar to the curves obtained with the previously evaluated carbonic anhydrase inhibitors.

Table 1 depicts the quantitative effect of the drug as a natriuretic agent. The average control urinary sodium excretion was 44 milliequivalents per 24 hours. Following the oral administration of 1,000 mg. twice daily, there was an increase in sodium excretion of 30 milliequivalents per 24 hours ($p<.001$). The 2,000 mg. single oral dose effected an increase sodium excretion of 28 milliequivalents per 24 hours ($p<.001$), and with the 4,000 mg. dose there was an increased sodium excretion of 31 milliequivalents per 24 hours ($p<.001$). The natriuretic potency is somewhat less at threshold dosages than is acetazolamide (Diamox), but Student's "t" test shows no significant difference between these two compounds in the augmentation of urinary sodium excretion.

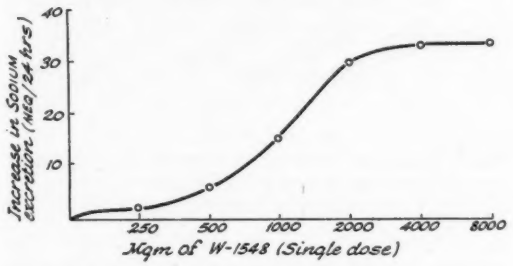

Fig. 2. Dosage response curve for sulocarbilate (W-1548-1) indicates the effective dose is 2,000 mg. or more.

TABLE 1.—*The Effect of Sulocarbilate Orally on the Excretion of Sodium.*

Patient	Excretion Control*	Increase After 1,000 mg. Twice Daily*	Increase After 2,000 mg. Once Daily*	Increase After 4,000 mg. Once Daily*
1	40	43	10	20
2	42	24	47	22
3	45	27	10	81
4	43	22	25	15
5	48	10	26	18
6	50	38	20	10
7	42	45	40	24
8	44	50	43	45
9	46	20	20	38
10	43	22	35	34
Average	44	30	28	31
p†		<.001	<.001	<.001
‡p			§n.s.	§n.s.

*All values expressed as milliequivalents of sodium per 24 hours.

†p value derived from Student's "t" test between control and drug values.

‡p value derived from "t" test between the various drug values.

§n.s. = not significant.

The electrolyte excretion pattern (fig. 3) following the oral administration of 2,000 mg. twice daily effected the changes characteristic of carbonic anhydrase inhibitors. The rate of sodium and potassium excretion showed roughly equivalent increases of about three times control level. This returned to near control levels after the second 2 hour period. Ammonia was suppressed initially but subsequently approximated control levels. There was a slight transitory increase in chloride excretion (not significant). There was a marked increase in bicarbonate excretion following both doses. Phosphate excretion was sup-

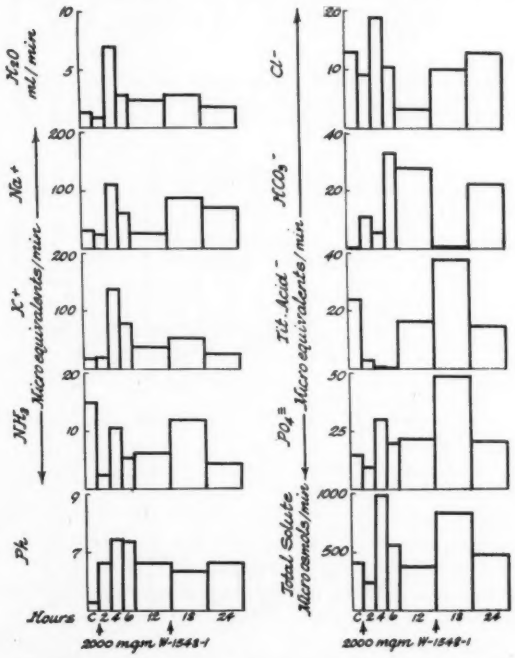

Fig. 3. Electrolyte excretion pattern following oral administration of 2,000 mg. of sulocarbilate (W-1548-1) twice daily. There is a significant increase in sodium, potassium, and bicarbonate excretion.

pressed initially but showed a marked rise following administration of the second dose. These data may be compared with a normal electrolyte excretion pattern (fig. 4).

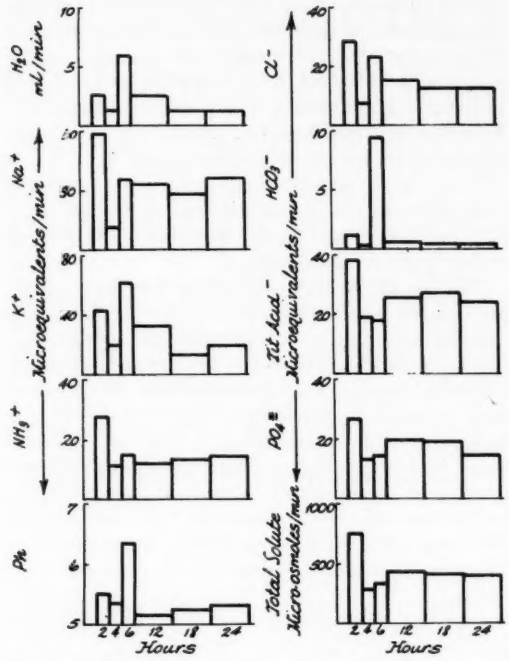

Fig. 4. Control electrolyte excretion pattern.

DISCUSSION

The use of carbonic anhydrase inhibitors, as oral diuretics, has found clinical application in the treatment of mild edema due to numerous causes. The mechanism of the diuretic effect is the inhibition of

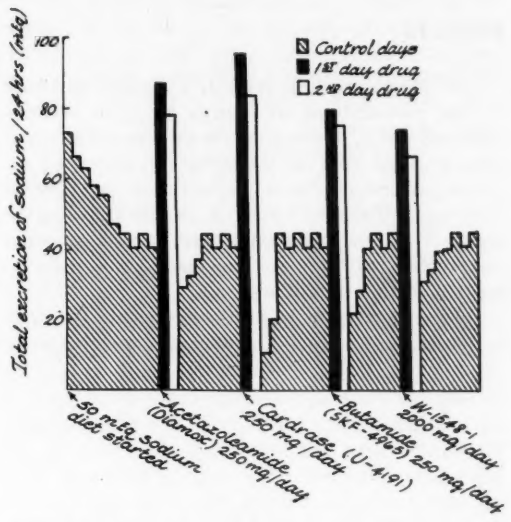

Fig. 5. Composite bar graph to illustrate changes in sodium excretion due to carbonic anhydrase inhibitors. The natriuretic effects are roughly equal and are of transitory duration.

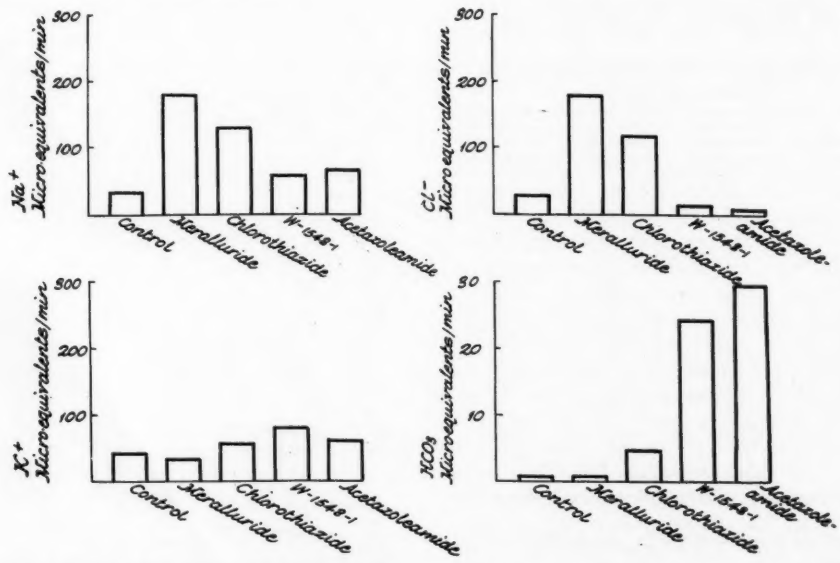

Fig. 6. Graph to illustrate the electrolyte excretion pattern of mercurials, carbonic anhydrase inhibitors, and chlorothiazide. Comparison of these patterns suggests that chlorothiazide utilizes another enzyme mechanism in effecting its diuretic response.

the carbonic anhydrase system at the level of the distal tubule in the kidney. This enzyme system is responsible for the exchange of intracellular hydrogen ions with the intratubular sodium which results in acidification of the urine. Inhibition of this process thus increases the excretion rate of sodium and potassium followed by an obligatory loss of water. Glomerular filtration rate and renal blood flow are not increased. Thus, the diuretic response is related primarily to the tubular effect of these compounds.

In preclinical studies concerning acute toxicity, the oral median lethal dose was found to be 15,000 mg. per kilogram of body weight in rats and 8,000 mg. per kilogram in dogs. The chronic administration to rats produced no adverse effect on growth, hematology, or fertility. At autopsy, no gross or microscopic disease was encountered. The over-all effects of the drug were similar to acetazoleamide, but the dosage required was approximately five to ten times greater than that of acetazoleamide.

The diuretic potency of this drug is slightly less than that of acetazoleamide (fig. 5). However, the difference in diuretic potency between this and other carbonic anhydrase inhibitors is not statistically significant. Our data indicate that optimum diuretic response to this drug is produced in a dose of 2,000 mg. given as a single oral dose and 1,000 mg. of this agent given twice daily. No toxic reactions have been observed with sulocarbilate. As is characteristic of the dosage response of carbonic anhydrase inhibitors, further increments in dosage above 2,000 mg. daily produce no augmentation in diuretic effect. In contrast the diuretic response to oral chlormerodrin (Neohydrin) or meralluride (Mercuhydrin) given parenterally continues to increase when doses are given which are greater than those generally employed clinically.

The electrolyte excretion patterns of the three presently most popular diuretic agents reflect differences that are worthy of comment (fig. 6). With mercurial diuretics there is primarily an increase in sodium, chloride, and water excretion. Inhibition of carbonic anhydrase effects an increase in sodium, potassium, and bicarbonate excretion. Chlorothiazide (Diuril) causes an increase in sodium and chloride excretion roughly equivalent to that seen with mercurials. However, there is also an increase in potassium and bicarbonate excretion of moderate degree. The reasons for the difference seen with chlorothiazide are presently not apparent. It would seem that either a new enzyme system is being utilized or else chlorothiazide competes with either one or both of the previously mentioned enzymes. Studies are presently being carried out further to define these differences.

SUMMARY AND CONCLUSIONS

The diuretic response to a carbonic anhydrase inhibitor, sulocarbilate, has been studied. There is no significant difference in its ability to increase water and sodium excretion as compared to acetazoleamide (Diamox). The optimum diuretic dose is approximately 2,000 mg. daily. There is no significant difference in diuretic response between 2,000 mg. as a single dose and 1,000 mg. given twice daily. No toxicity was encountered in the bioassay of this agent.

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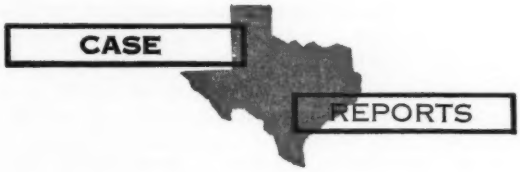

Tuberous Sclerosis And Hirsutism In a Negress

M. JEANNE FAIRWEATHER, M.D.; D. L. GALINDO, M.D.; and ROBERT C. HARDY, M.D.

San Antonio, Texas

SINCE the first description of tuberous sclerosis by Bourneville and Brissaud (1880) as a disease characterized by the triad adenoma sebaceum, convulsions, and mental retardation, others have described additional aspects of this somewhat rare disease. Von Recklinghausen (1862) previously had described sclerotic plaques in the brain and rhabdomyoma in the heart of a newborn infant. Bourneville (1880) added idiocy associated with tuberous changes in the cortex; Pringle (1890) described adenoma sebaceum and nevoid conditions of the skin;

van der Hoeve (1921), retinal phakomas; Marcus (1924), the skull lesions; Ackerman (1938), femoral cysts; Yakoleo (1939), calcium deposits and brain tumors; Berland (1953) and Dickerson (1955), cyst-like lesions in the phalanges with irregular thickening and cysts of the cortex of the metatarsal and metacarpal bones.

Dr. M. Jeanne Fairweather and her co-authors submitted this paper from the sections on medicine and surgery of Robert B. Green Memorial Hospital and San Antonio Division of the University of Texas Postgraduate School of Medicine.

This case of tuberous sclerosis in a 20 year old Negro girl demonstrated the typical findings of the condition as reported in the literature as well as several unusual aspects, including hirsutism.

The skin lesions associated with tuberous sclerosis may be present at birth but usually do not appear until the child is 3 to 6 years of age. The most common and typical form is multiple warty nodules in a butterfly distribution on the side of the nose, nasolabial folds, and cheeks. They vary in color from pale to dark brown. Other forms are pedunculated skin polyps, pigmented nevi and warts, café au lait spots, and Vorner's anemic (vitiligo) nevi patches in the skin. The facial adenoma sebaceum alone may be found in relatives of patients suffering from the disease.

Small angiofibromatous growths (subungual angiofibroma), may be found at the margin of the fingernails. At times peau de chagrin—a rough, irregular, elevated, white or pink, thickened area of skin—may be found over the lower part of the back.

Epileptiform attacks are common and may be minor, major, or jacksonian, the onset being from 1 year of age to puberty. There may be but one convulsion in a lifetime, or many. Temper outbursts, vertiginous attacks, various forms of psychosis, or hallucinations may be noted. The patient may appear normal at birth but be late in crawling, walking, talking, and sitting. In abortive forms, there is no mental deficiency. Speech disturbances are common.

Examination of the brain reveals hard, white nodules on the surface of the cortex which are smooth and project above the surface of the neighboring cortex. They may involve only a portion of one convolution or they may extend to a major portion of a hemisphere. Nodules appear in subcortical gray matter, white matter, and basal ganglions; small nodules called "candle gutterings" project into lateral ventricular cavities. Sclerotic nodules may be found in the brain stem, cord, and cerebellum. There may be vascular hyperplasia and actual angiomatous malformations. Occasionally, large tumors described as spongioblastomas or neurospongioblastomas may be found. Dickerson (1955) thought that at least 50 per cent of patients with tuberous sclerosis show intracranial calcifications in the region of basal ganglions, and a small number show them in the dentate nucleus of the cerebellum. Hemiplegia, diplegia, and athetosis may occur. There are no special electroencephalographic patterns although many tracings are abnormal. Nodular areas in the region of the hypothalamus are believed to account for bouts of fever which may occur.

Retinal tumors may be the sole indications of tuberous sclerosis. They extend through the vitreous into which loosened and detached parts of the tumor may fall. They are correctly described as neurocytophakoma retinae. They appear as yellowish to rosy spots with or without pigmentation, circumscribed nevoid lesions, and sometimes resembling retinitis pigmentosa. Choked disk and other neurological signs occur with development of large intracerebral tumors.

Renal tumors occur in 66 to 80 per cent of cases. They involve the cortex and are mixed as sarcomas, adenomas, fibromas, angiomas, and harmatomas. The radiographic appearance of the kidneys is similar to polycystic kidneys; however, Beck noted a reticulated pattern in the kidney parenchyma, which he believed was due to a lipomatous component within the tumor. Cardiac tumors (rhabdomyomas) occur in 5 per cent. They are usually small and symptomless. A few cases of lesions appearing as miliary or cystic tuberculosis have been described.

Dickerson (1955) stated that 66 per cent have changes in the phalanges of the hands and feet. There are irregular wavy periosteal thickenings of the metatarsals and metacarpals. The areas appearing as cysts in the roentgenograms are not cysts but are specific fibrous replacement of bone encompassed by a proliferative process resulting in abundant steroid matrix. Periosteal changes have a predilection for the feet, cysts for the hands. It is observed that if phalangeal lesions are found, cranial plaques or intracranial calcifications will be present. Cysts also may be found in the vertebrae and long bones.

This disease is notorious for congenital malformations of the skull, ears, and face; high palate; cryptorchism; spina bifida; and extra digits. In formes frustes, brain lesions are minor, whereas in the full blown variant, the brain, face, eyes, skin, and internal organs all may be involved.

The following case illustrates the various typical aspects of this disease as well as several unusual features.

CASE REPORT

CASE 1.—A 20 year old Negro girl was first seen in the Robert B. Green Memorial Hospital emergency room February 13, 1957, with the chief complaint of blindness, convulsions, and vomiting, and was admitted to the hospital.

The patient was in good health until 1954 at which time she experienced occipital pain, stiff neck, and headache lasting a few days. One week later she fainted. In July, 1955, while working in a cotton field she became very hot, complained of severe headache, and suddenly became unconscious. The patient was examined by a physician who stated that she probably had poliomyelitis. She remained in bed 5 days and then returned to her work. About 1 year later, she began to complain of headaches, and her mother noted that she fumbled when reaching for something with her right hand. Shortly thereafter, she complained of diplopia. A diagnosis of brain tumor was made at that time. Loss of vision progressed, particularly in the left eye, and she complained of dizzy spells. The patient was seen by several physicians who told her family the patient had a brain tumor.

She began to lose weight, complained of dysuria, hematuria, passing "gravel," frontal headaches, and drowsiness. In October, 1956, approximately 2 years after the onset of her symptoms, she had a grand mal seizure accompanied by salivation and incontinence. The patient was seen at a large medical center where she was told that she had a brain tumor and a kidney tumor. Surgery was not suggested at that time. The patient returned home and again had grand mal seizures in January and February, 1957. The last seizure was accompanied by vomiting five times.

The patient's past history revealed that she was born at full term, but developed much slower than her siblings, walking and talking many months later. She stuttered from the time she started to talk. A "wartlike" eruption began to appear on her face at about 6 years of age. Hair appeared on her upper lip, chin, temporal regions, and breasts at age 16 years. Menstruation was normal. She was in the ninth grade at age 17 years, made fair grades, but found studying very difficult. The remainder of her history was insignificant.

The paternal great grandfather had wartlike lesions on his face and neck as did her paternal grandfather. Her father has large pedunculated nevi on his neck. The mother has areas of depigmentation of her skin. There were 12 siblings. Two sisters have areas of depigmentation; one sister has adenoma sebaceum on her face and has petit mal seizures (fig. 1).

Physical examination revealed a slim, confused, drowsy 20 year old Negro girl. Temperature was 99 F., pulse rate 80 per minute, respiration 20, and blood pressure 130/70. The pupils were round, responding sluggishly to light. The fundi displayed massive papilledema. The optic disks were white, and central choroidal atrophy was seen. There was no light perception and no nystagmus. Adenoma sebaceum was seen over the nose and cheeks. A fine mustache was noted as well as heavy growth of hair on the chin (fig. 2). There was no nuchal rigidity. The chest was clear to percussion and auscultation. The breasts were small, and there was long hair about the areola. The pulse rate was regular; no heart murmurs were noted. No abdominal masses or tenderness was palpated. Pelvic examination revealed a male distribution of pubic hair, a slightly enlarged clitoris, and mild vaginitis. The deep tendon reflexes were hyperactive, but equal.

Five days later, it was noted that there was bilateral horizontal nystagmus with the slow component laterally. There was also marked ulcerations of the left cornea. The upper lid was denuded. The anterior chamber was cloudy. It was impossible to see the fundus. The papilledema was subsiding on the right.

Laboratory studies revealed no unusual findings except for a glucose tolerance curve which gave the following

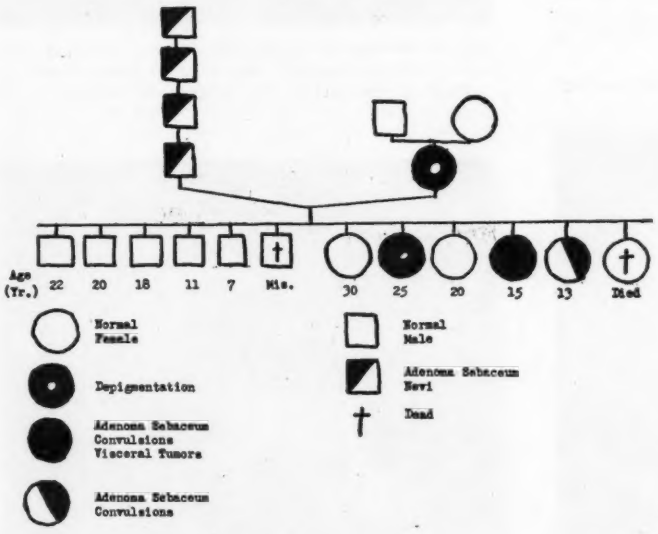

Fig. 1. Family history of a patient with tuberous sclerosis. The age of the 6 female siblings should read 30, 25, 15, 20, 13, Died.

Fig. 2. Face of the patient showing adenoma sebaceum in butterfly distribution over cheeks and nose, ulceration of the left eyelids, faint moustache, and chin hair.

results: fasting 78 mg. of glucose per 100 cc. of blood, ½ hour 90, 1 hour 94, 2 hours 68, 3 hours 68. Urinary 17-ketosteroid and corticoid excretions were normal, and a normal response to 60 units of ACTHAR Gel was observed.

Radiologic examinations revealed a mottled calcification 1 inch in diameter lying in the midline of the brain and 1 inch above the uppermost portion of the posterior clinoids. The sella tursica was enlarged; there was a small degree of erosion of the floor of the dorsum sella and increased gyral markings. The mottlings resembled those in Rathke's pouch tumor or craniopharyngeoma (fig. 3). An intravenous pyelogram revealed the left kidney to be normal; the right pelvis was enlarged and grossly distorted, dilated, irregular, and motheaten. A retrograde examination was done, and irregularity and deformity of the collecting system was seen in the right. Extravasation of radiopaque material in the subcapsular portion of the superior pole was noted. The inferior calyces were displaced medially, and there was marked displacement of the middle calyx inferiorly (fig. 4). A cerebral arteriogram revealed that the anterior cerebrals on the right filled partially and were displaced laterally (fig. 5). Small radiolucent defects in the dorsal cortex of the midshaft of the middle phalanx of the ring finger, also in the angular minor, capitate, and distal shaft of the proximal phalanx of the middle finger of the right hand, were seen. This defect also was found in the distal portion of the proximal phalanx of the middle finger and in the base of the second and fourth metacarpals. There were wavy irregularities in the periosteal outline and small oblong radiolucent defects in the inferolateral cortex of the midshaft of the fourth metatarsals.

The pathologist reported a biopsy of the skin lesions to be consistent with adenoma sebaceum.

The patient complained of increasingly severe headache no longer controlled by codeine. The diagnosis of tuberous sclerosis was made, and it was believed she might also have a craniopharyngoma. On the twenty-first hospital day, the patient was believed to be deteriorating rapidly, and it was decided that surgery for relief of intracranial pressure was essential.

Under Pentothal and nitrous oxygen anesthesia a craniotomy with aspiration of the craniopharyngeal cyst was performed. No tumor could be visualized. The patient was returned to the recovery room in poor condition. Skull roentgenograms made immediately after surgery revealed a left central bone flap and a small amount of air in the ventricle. The calcified shadow previously mentioned was still present. The patient did not respond well following surgery and died 5 hours postoperatively.

Necropsy Results.—At necropsy, the following outstanding gross and microscopic lesions were found:

Heart: Small, irregular, white-yellow adipose tissue masses of up to 2.0 cm. in diameter in the myocardium and particularly in the interventricular septum. The masses were made up of lobules of mature adipose tissue.

Lungs: The pleural surfaces throughout both lungs presented small blue-gray and purple cystic structures measuring up to 1.5 cm. in diameter and filled with clear watery or serosanguineous fluid.

Kidneys: The upper pole of the right kidney contained a large retention cyst measuring 6.0 cm. in diameter and containing yellow-green fluid. On section there were soft, irregular, lobular, gray-brown "whorled" masses measuring

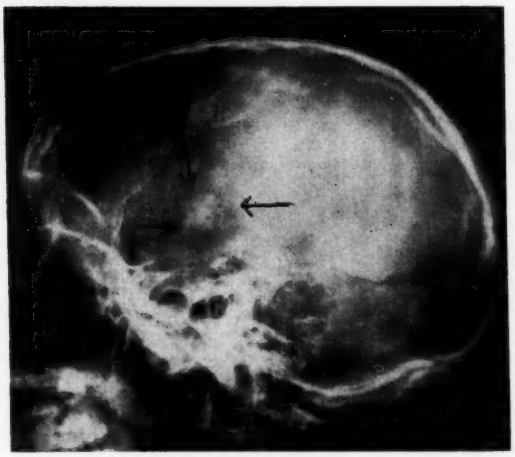

Fig. 3. Roentgenogram of skull showing mottled calcifications above the posterior clinoids, erosion of the floor of the dorsum sella, and increased gyral markings.

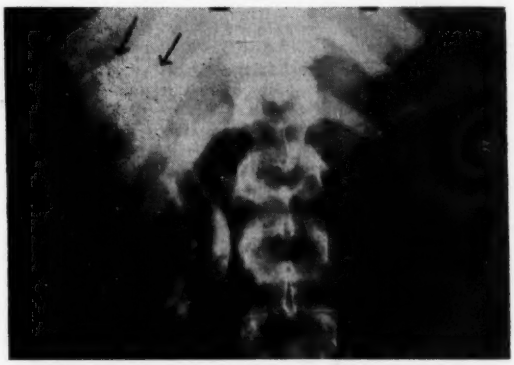

Fig. 4. Retrograde pyelogram showing enlarged right kidney, displacement of calyces, and extravasation of radiopaque material in the subcapsular portion of the superior pole.

Fig. 5. Cerebral arteriogram revealing partially filled and laterally displaced right anterior cerebrals.

up to 6.0 cm. in diameter. The masses consisted of sheets of interlacing mature fibromuscular fibrils intermixed with fibroadipose tissue and fibrovascular elements. The left kidney was essentially the same as the right, but the tumor masses were smaller.

Thyroid: Several small irregular tumors appeared on the surface and consisted of well differentiated Hürthle cells supported by lobules of usual thyroid acini.

Skull and brain: In the region of the sella tursica there was a fragmented cystic mass partially filled with clotted blood and measuring 2.0 cm. in diameter. The mass was composed of a fragmented squamous epithelial lining. The cerebral and cerebellar hemispheres were slightly asymmetrical, distorted by multiple, semifirm, doughy, focally protruding masses with a "potato-like" consistency measuring up to 3.0 cm. in diameter (fig. 6). On multiple sections, the white and gray matter relationships were distorted by the "potato" consistency tumors which obliterated and flattened out the gray matter at various points of the cortex. The left anterior leg of the lateral ventricle was occupied by a "potato" consistency tumor 6.0 cm. in diameter at the widest point. It compressed and obliterated the nuclei in the corresponding basal ganglions and the corpus callosum. It extended across the midline and compressed the medial aspects of the right lateral ventricle. In the midportions of the tumor there were extensive cartilaginous and calcific deposits. In the tumor described in the ventricle there were bundles, sheaths, and interlacing groups of spindle, oblong, and oval large nerve cells with pleomorphic, vesicular, and hyperchromatic nuclei and densely eosinophilic staining cytoplasm. The supporting glial tissue was composed of fibrillar fragments. Part of the tumor was distorted by calcific deposits and large pools of recent and old extravasated blood. The tumor was morphologically characteristic of an astrocytoma.

The terminal events in the patient were hemorrhage into astrocytoma, minimal subarachnoid hemorrhage, diffuse pulmonary intra-alveolar hemorrhage, acute pulmonary edema, and focal atelectasis. Microscopically, the tumors presented were as follows: (1) angiolipofibroma of the kidney, (2) lipoma of the heart and lymph nodes, (3) astrocytoma of the left cerebral hemisphere, (4) "diffuse gliosis" or potato consistency masses of the left cerebral hemisphere, (5) supracellar cyst and epidermal inclusion cyst of the pituitary.

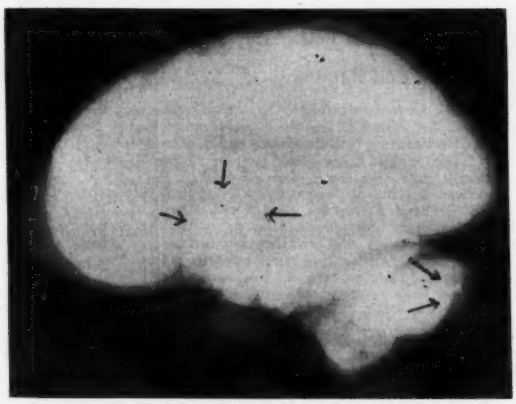

Fig. 6. Roentgenogram of the brain at autopsy showing asymmetrical cerebral and cerebellar hemispheres distorted by protruding masses with "potato-like" consistency.

DISCUSSION

A brief discussion of tuberous sclerosis and a case history with autopsy findings in a 20 year old Negro girl have been presented. This patient demonstrated all the typical findings of tuberous sclerosis as well as several unusual aspects, namely, that she was a Negro, that her seizures started later than most, and that she had become hirsute. No other case of tuberous sclerosis with hirsutism was found to be reported in the literature.

We wish to extend our appreciation to Dr. Herman S. Wigodsky, medical director, Robert B. Green Memorial Hospital, for his excellent photography and valuable assistance with this manuscript.

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Unmarried Women Develop Most Breast Cancer

Brothers, sisters, and parents of persons with stomach and breast cancer have a higher rate of cancer of the same type than does the general population, reported Clarence P. Oliver, Ph.D., zoology research director, University of Texas, Austin, from family history studies he is making. Dr. Dudley Jackson, Sr., San Antonio, is cooperating with Dr. Oliver in his genetic studies of cancer families.

Dr. Oliver is trying to determine what part heredity plays in causing cancer by collecting family histories of cancer patients. He reported that studies of family pedigrees indicate that susceptibility to cancer is inherited in certain families. Two major difficulties in collecting the histories of cancer families are, first, some persons refuse to give information about cancer in their family because they fear their family might suffer economically as a consequence, and, second, tracing the hereditary cancer line was difficult because only 71 per cent of cancer victims have had offsprings.

Unmarried women face a much greater possibility of having cancer than do married women, Dr. Oliver pointed out. Childless married women face a greater hazard of cancer than married women with children. Mothers who breast-feed their children have the lowest rate of breast cancer development, Dr. Oliver said.

MEDICAL NEWS

★ Coming Meetings

Texas Medical Association, Houston, April 19-22, 1958 (Executive Council, Jan. 19, 1958). Dr. Denton Kerr, Houston, Pres.; Mr. C. Lincoln Williston, 1801 North Lamar Blvd., Austin, Executive Secy.

American Medical Association, San Francisco, June 23-27, 1958. Dr. David B. Allman, Atlantic City, Pres.; Dr. George F. Lull, 535 North Dearborn, Chicago 10, Secy.

Current Meetings

APRIL

American Academy of Obstetricians and Gynecologists, Los Angeles, April 21-23, 1958. Dr. R. Gordon Douglas, New York, Pres.; Dr. John C. Ullery, Ohio State University Hospital, Columbus, Secy.

American Academy of Pediatrics, New York, April 21-23, 1958. Dr. Stewart H. Clifford, Brookline, Mass., Pres.; Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill., Secy.

American Association of Genito-Urinary Surgeons, Edgewater Park, Miss., April 23-25, 1958. Dr. George F. Cahill, New York, Pres.; Dr. W. J. Engel, 2 East 54th, New York 22, Secy.

American College of Allergists, Atlantic City, April 20-25, 1958. Dr. Oval R. Withers, Kansas City, Pres.; Dr. Giles A. Koelsche, Mayo Clinic, Rochester, Minn., Secy.

American College of Physicians, Atlantic City, April 28-May 2, 1958. Dr. Richard A. Kern, Philadelphia, Pres.; Mr. E. R. Loveland, 4200 Pine, Philadelphia 4, Secy.

American Surgical Association, New York, April 16-18, 1958. Dr. John H. Mulholland, New York, Pres.; Dr. William Altemeier, Christian R. Holmes Hospital 19, Cincinnati, Secy.

American Urological Association, New Orleans, April 28-May 1, 1958. Dr. William J. Baker, Chicago, Pres.; Dr. Samuel L. Raines, 188 S. Bellevue Blvd., Memphis, Tenn., Secy.

Association of American Physicians and Surgeons, San Francisco, April 10-19, 1958. Dr. Cyrus W. Anderson, Denver, Pres.; Mr. Harry E. Northam, 185 N. Wabash Ave., Chicago 1, Executive Secy.

Southwestern Society of Nuclear Medicine, Dalals, April 12-13, 1958. Dr. Jack G. S. Maxfield, Dallas, Pres.; Dr. J. R. Maxfield, Jr., 311 Medical Arts Bldg., Dallas, Secy.

United States-Mexico Border Public Health Association, Hermosillo, Sonora, Mexico, April, 1958. Malcolm H. Merrill, Berkeley, Calif., Pres.; Dr. Sidney B. Clark, 243 U. S. Court House, El Paso, Secy.

Texas Air-Medics Association, Houston, April 20-21, 1958. Dr. W. D. Marrs, Fort Worth, Pres.; Dr. C. F. Miller, P. O. Box 1338, Waco, Secy.

Texas Chapter, American Association of Public Health Physicians, Houston, April 20, 1958. Dr. Roy G. Reed, Victoria, Pres.; Dr. A. R. Doane, 410 E. 5th, Austin, Secy.

Texas Chapter, American College of Chest Physicians, Houston, April 20, 1958. Dr. John A. Wiggins, Fort Worth, Pres.; Dr. H. M. Anderson, 224 E. Harris, San Angelo, Secy.

Texas Dermatological Society, Houston, April 20-29. Dr. Charles D. Stewart, Corpus Christi, Pres.; Dr. E. N. Walsh, 1310 Medical Arts Bldg., Fort Worth, Secy.

Texas Diabetes Association, Houston, April 20, 1958. Dr. H. T. Engelhardt, Houston, Pres.; Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Secy.

Texas Geriatrics Society, Houston, April 21, 1958. Dr. Martin S. Buehler, Dallas, Pres.; Dr. J. A. Armstrong, 3810 Swiss Ave., Dallas, Secy.

Texas Heart Association, Houston, April 20, 1958. Dr. James A. Greene, Houston, Pres.; Mr. Edgar M. Brown, 404 Jesse H. Jones Library Bldg., Texas Medical Center, Houston 25, Executive Director.

Texas Industrial Medical Association, Houston, April 20, 1958. Dr. Robert J. Potts, Houston, Pres.; Dr. Robert A. Wise, Box 2180, Houston, Secy.

Texas Neurophychiatric Association, Houston, April 20, 1958. Dr. Bruce H. Beard, Fort Worth, Pres.; Dr. Clarence S. Hoekstra, 8215 Westchester Drive, Dallas, Secy.

Texas Ophthalmological Association, Houston, April 22, 1958. Dr. Thomas J. Vanzant, Houston, Pres.; Dr. Louis Daily, Medical Arts Bldg., Dallas, Secy.

Texas Orthopedic Association, Houston, April 21, 1958. Dr. Margaret Watkins, Dallas, Pres.; Dr. B. C. Halley, Jr., 524 Doctors Building, Dallas, Secy.

Texas Physical Medicine and Rehabilitation Society, Houston, April, 1958. Dr. Odon F. von Werssowetz, Gonzales, Pres.; Dr. Edward M. Krusen, Baylor Hospital, Dallas, Secy.

Texas Society of Anesthesiologists, Houston, April 20, 1958. Dr. J. D. McCulley, Houston, Pres.; Dr. Randle J. Brady, 3317 Binz, Houston, Secy.

Texas Society of Gastroenterologists and Proctologists, Houston, April 20, 1958. Dr. Tate Miller, Dallas, Pres.; Dr. O. P. Griffin, 1101 Medical Arts Bldg., Fort Worth, Secy.

Texas Society of Pathologists, Houston, April 22, 1958. Dr. John H. Childers, Galveston, Pres.; Dr. Mervin H. Grossman, Box 57, Dallas, Secy.

Texas Society of Plastic Surgeons, Houston, April 19, 1958. Dr. Truman G. Blocker, Galveston, Pres.; Dr. Steve R. Lewis, University of Texas Medical Branch, Galveston, Secy.

Texas Surgical Society, El Paso, April 6-7, 1958. Dr. J. Peyton Barnes, Houston, Pres.; Dr. G. V. Brindley, Jr., Scott and White Clinic, Temple, Secy.

Texas Traumatic Surgical Society, Houston, April 20, 1958. Dr. Russell Holt, El Paso, Pres.; Dr. W. E. Crump, 1300 Eighth, Wichita Falls, Secy.

State Tumor Conference, Wichita Falls, April 12, 1958. Dr. Edwin C. Bebb, 500 Broad St., Wichita Falls, Director.

Texas State Board of Examiners in Basic Sciences, April, 1958. Henry B. Hardt, Ph.D., Fort Worth, Pres.; Mrs. Betty J. Ratliff, Chief Clerk, 303 East Seventh, Austin.

MAY

American Association for Thoracic Surgery, Boston, May 16-18, 1958. Dr. Brian Blades, Washington, D. C., Pres.; Dr. Hiram T. Langston, 600 S. Kingshighway, St. Louis 10, Mo., Secy.

American Gastro-Enterological Association, Washington, D. C., May 30-31, 1958. Dr. F. J. Ingelfinger, 65 E. Newton, Boston 18, Secy.

American Gynecological Society, Asheville, N. C., May 19-21, 1958. Dr. Howard C. Taylor, Jr., New York City, Pres.; Dr. A. A. Marchetti, 3800 Reservoir Rd., N.W., Washington 7, D. C., Secy.

American Laryngological, Rhinological, and Otological Society, San Francisco, May 21-23, 1958. No 1957 meeting. Dr. Lawrence R. Boies, Minneapolis, Pres.; Dr. C. S. Nash, 277 Alexander, Rochester 7, N. Y., Secy.

American Ophthalmological Society, White Sulphur Springs, W. Va., May 28-30, 1958. Dr. Walter S. Atkinson, Watertown, N. Y., Pres.; Dr. M. C. Wheeler, 30 W. 59th, New York 19, Secy.

American Pediatric Society, Atlantic City, N. J., May 8-9, 1958. Dr. A. Ashley Weech, Cincinnati, Pres.; Dr. A. C. McGuinness, Room 1036, 2800 Quebec St., N.W., Washington 8, D. C., Secy.

American Psychiatric Association, San Francisco, May 12-16, 1958. Dr. Harry C. Solomon, Boston 15, Pres.; Dr. William Malamud, 80 E. Concord, Boston 18, Secy.

National Tuberculosis Association, Philadelphia, May 18-23, 1958. Dr. William M. Morgan, New York, Pres.; Mrs. Wallace B. White, 1790 Broadway, New York 19, Secy.

Texas Hospital Association, Dallas, May 5-8, 1958. Bolton Boone, D.D., Dallas, Pres.; Mr. O. Ray Hurst, Executive Director, 2208 Main, Dallas, Secy.

National and Regional

American Academy of Allergy. Dr. Carl E. Arbesman, Buffalo, N. Y., Pres.; Dr. Francis C. Lowell, 65 E. Newton, Boston, Secy.

American Academy of Dermatology and Syphilology, Chicago, Dec. 6-11, 1958. Dr. E. R. Seale, Houston, Pres.; Dr. R. R. Kierland, Mayo Clinic, Rochester, Minn., Secy.

American Academy of General Practice, San Francisco, April 6-9, 1959. Dr. Holland T. Jackson, Fort Worth, Pres.; Mr. Mac F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Executive Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 12-17, 1958. Dr. Leroy A. Schall, Boston, Pres.; Dr. W. L. Benedict, 15 Second St. S.W., Rochester, Minn., Secy.

American Association for Maternal and Infant Health, 1960. Mr. Howard I. Wells, Jr., 116 S. Michigan Ave., Chicago 3, Executive Secy.

American Association of Obstetricians and Gynecologists. Dr. William F. Mengert, Chicago, Pres.; Dr. E. Stewart Taylor, University of Colorado School of Medicine, 4200 E. 9th, Denver, Secy.

American Cancer Society, New York, Oct. 29-31, 1958. Dr. L. T. Coggeshall, Chicago, Pres.; M. R. Runyon, 521 W. 57th, New York, Executive Vice-Pres.

American College of Chest Physicians, San Francisco, June 18-22, 1958. Dr. Burgess L. Gordon, Philadelphia, Pres.; Mr. Murray Kornfeld, 112 E. Chestnut, Chicago 11, Executive Secy.

American College of Gastroenterology, New Orleans, Oct. 20-28, 1958. Dr. C. Wilmer Wirts, Philadelphia, Pres.; Mr. Daniel Weiss, 33 West 60th, New York 23, Executive Director.

American College of Radiology. Dr. Vincent M. Archer, Charlottsville, Va., Pres.; Mr. W. C. Stronach, 20 N. Wacker Dr., Chicago 6, Executive Director.

American College of Surgeons, Chicago, Oct. 6-10, 1958. Dr. William L. Estes, Bethlehem, Pa., Pres.; Dr. Michael L. Mason, 40 E. Erie, Chicago 11, Secy.

American Congress of Physical Medicine and Rehabilitation, Philadelphia, Aug. 24-29, 1958. Dr. Donald L. Rose, Kansas City, Kan., Pres.; Dorothea C. Augustin, 30 N. Michigan Ave., Chicago, Executive Secretary.

American Dermatological Association, Sun Valley, June 4-8, 1958. Dr. Clark W. Finnerud, Chicago, Pres.; Dr. Hamilton Montgomery, 200 1st, S.W., Rochester, Minn., Secy.

American Heart Association, San Francisco, Oct. 24-28, 1958. Dr. Robert W. Wilkins, New York, Pres.; Mr. John D. Brundage, 44 E. 23rd, New York 10, Secy.

American Hospital Association, Chicago, Aug. 18-21, 1958. Mr. Tol Terrell, San Angelo, Pres.; Dr. Edwin L. Crosby, 18 E. Division, Chicago 10, Executive Director.

American Neurological Association, Atlantic City, June 16-18, 1958. Dr. Israel S. Wechsler, New York, Pres.; Dr. Charles Rupp, 133 S. 36th, Philadelphia 4, Secy.

American Orthopedic Association, Hot Springs, Va., June 24-27, 1958. Dr. George O. Eaton, Baltimore, Pres.; Dr. Harold A. Safield, 715 Lake St., Oak Park, Ill.

American Proctologic Society, Los Angeles, June 29-July 3, 1958. Dr. Julius E Linn, Birmingham, Pres.; Dr. Norman D. Nigro, 10 Peterboro, Detroit 1, Secy.

American Public Health Association, St. Louis, Oct. 27-31, 1958. Roy J. Morton, Oak Ridge, Tenn., Pres.; Dr. Berwyn F. Mattison, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists, Pittsburgh, Pa., Oct. 19-24, 1958. Dr. Ralph S. Sappenfield, Miami, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph, Chicago 1, Secy.

American Society of Clinical Pathologists, Chicago, Nov. 1-8, 1958. Dr. Harry P. Smith, New York, Pres.; Dr. Clyde G. Culbertson, 1040 W. Michigan, Indianapolis 7, Secy.

International College of Surgeons, U. S. Chapter. Dr. Curtice Rosser, Dallas, Pres.; Dr. Karl Meyer, 1516 Lake Shore Dr., Chicago, Secy.

Radiological Society of North America, Chicago, Nov. 16-21, 1958. Dr. Leo G. Rigler, Los Angeles, Pres.; Dr. Donald S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

Southern Medical Association, New Orleans, Nov. 3-6, 1958. Dr. W. Kelly West, Oklahoma City, Pres.; Mr. V. O. Foster, 1020 Empire Bldg., Birmingham 3, Secy.

Southern Surgical Association, Boca Raton, Fla., Dec. 9-11, 1958. Dr. James D. Rives, New Orleans, Pres.; Dr. George G. Finney, 2947 St. Paul, Baltimore 18, Secy.

Southern Psychiatric Association, Tennessee, October, 1958. Dr. Frank Luton, Nashville, Pres.; Dr. Iverson O. Brownell, Greenville, S. C., Secy.

Southwest Allergy Forum, Houston, 1959. Dr. Homer E. Prince, 808 Carolina, Houston, Pres. and Secy.

Southwest Regional Cancer Conference, Fort Worth, September, 1958. Dr. O. J. Wollenman, Jr., Fort Worth, Chm.; Mrs. Ira Frances Ball, 264 W. 11th, Fort Worth, Secy.

Southwestern Medical Association, Tucson, Ariz., Oct. 23-25, 1958. Dr. Louis G. Jekel, Phoenix, Pres.; Dr. Russell L. Deter, 1501 Arizona, El Paso, Secy.

Southwestern Surgical Congress. Dr. Kenneth C. Sawyer, Denver, Pres.; Dr. C. M. O'Leary, 207 Plaza Court Bldg., Oklahoma City, Secy.

Tri-State Medical Assembly, Shreveport, Sept. 18, 1958. Dr. James Gatline, Camden, Ark., Pres.; Dr. Jason Sanders, Sanders Clinic, Kings Highway, Shreveport, La., Secy.

State

Private Clinics and Hospitals Association of Texas, Dallas, Dec. 14-15, 1958. Dr. Joe Shepperd, Burnet, Pres.; Mr. Henry E. Taylor, Medical Arts Building, Dallas, Executive Secy.

Texas Academy of General Practice, San Antonio, Sept. 22-24, 1958. Dr. G. W. Cleveland, Austin, Pres.; Mr. Donald C. Jackson, 1905 N. Lamar, Austin, Executive Secy.

Texas Academy of Internal Medicine, Galveston, Dec. 6-7, 1958. Dr. A. W. Harris, Dallas, Pres.; Dr. Hugo T. Engelhardt, 1216 Main, Houston, Secy. Meetings restricted to members.

Texas Association of Blood Banks, Austin, February, 1959. Dr. Louis J. Manhoff, Jr., San Antonio, Pres.; L. Ruth Guy, Ph.D., 725 Doctors Building, Temple.

Texas Association of Obstetricians and Gynecologists, Fort Worth, February, 1959. Dr. Jesson L. Stowe, El Paso, Pres.; Dr. Oran V. Prejean, 4317 Oak Lawn, Dallas, Secy.

Texas Association for Mental Health. Dr. Virginia Love, Houston, Pres.; Mr. John Lane, 2510 San Antonio, Austin, Executive Director.

Texas Club of Internists. Dr. D. D. Warren, Waco, Pres.; Dr. L. C. Carter, 2600 Procter, Port Arthur, Secy.

Texas Division, American Cancer Society, Austin, Dec. 4, 1958. Dr. J. Layton Cochran, San Antonio, Pres.; Mr. Curt W. Reimann, 5014 Bull Creek Rd., Austin 3, Executive Director.

Texas Pediatric Society, Fort Worth, Oct. 17-18, 1958. Dr. T. J. McElhenney, Austin, Pres.; Dr. James N. Walker, 5216 W. Freeway, Fort Worth, Secy.

Texas Proctologic Society, 1959. Dr. Hugh Beaton, Fort Worth, Pres.; Dr. C. P. Hardwicke, 920 E. 32nd, Austin, Secy.

Texas Public Health Association. Mr. J. N. Murphy, Jr., Austin, Pres.; Mr. Wayne Garrett, City Health Department, Fort Worth, Executive Secy.

Texas Radiological Society, Dallas, Jan. 30-31, 1959. Dr. R. F. Wertz, Amarillo, Pres.; Dr. J. E. Miller, 6407 Forest Lane, Dallas 30, Secy.

Texas Rheumatism Association. Dr. W. W. Bondurant, San Antonio, Pres.; Dr. Morris Horn, 3707 Gaston, Dallas, Secy.

Texas Tuberculosis Association. Dr. Howard T. Barkley, Houston, Pres.; Miss Pansy Nichols, P. O. Box 6158, Austin 21, Executive Director.

Texas Society of Ophthalmology and Otolaryngology, Houston, Dec. 5-6, 1958. Dr. Owen R. O'Neill, Paris, Pres.; Dr. Edwin G. Grafton, 4319 Oak Lawn, Dallas, Secy.

Texas Urological Society, San Antonio, January, 1959. Dr. Charles Hooks, Galveston, Pres.; Dr. Charles Hulse, 636 Moore Building, San Antonio.

District

First District Society. Dr. W. A. Jones, El Paso, Pres.; Dr. E. S. Crossett, 309 Medical Arts Bldg., El Paso, Secy.

Second District Society, Lamesa. Dr. J. Vernon McKay, Lamesa, Pres.; Dr. Noble L. Rumbo, Box D, O'Donnell, Secy.

Third District Society. Dr. William Klingensmith, Amarillo, Pres.; Dr. H. Fred Johnson, 2308 W. Eighth, Amarillo, Secy.

Fourth District Society, San Angelo, 1958. Dr. Fred D. Spencer, Brownwood, Pres.; Dr. W. Lacey Smith, 111 E. Harris, San Angelo, Secy.

Fifth and Sixth Districts Society, Corpus Christi, 1958. Dr. Foy Moody, Corpus Christi, Pres.; Dr. Thelma Frank, 129 Rainbow Lane, Corpus Christi, Secy.

Seventh District Society. Dr. Robert N. Snider, Austin, Pres.; Dr. Richard Lucas, 502 W. 13, Austin, Secy.

Eighth District Society, Galveston, October, 1958. Dr. John H. Childers, Galveston, Pres.; Dr. M. Warren Hardwick, 839 E. Mulberry, Angleton, Secy.

Ninth District Society, Conroe. Dr. Eugene M. Addison, Huntsville, Pres.; Dr. James H. Sammons, Highlands, Secy.

Tenth District Society. Dr. B. F. Pace, Beaumont, Pres.; Dr. W. J. Poshataske, Silsbee, Secy.

Eleventh District Society. Dr. George M. Hilliard, Jacksonville, Pres.; Dr. Phillip W. Taylor, 833 S. Beckham, Tyler, Secy.

Twelfth District Society. Dr. George C. Bryant, Waco, Pres.; Dr. J. T. Archer, Jr., Meridian, Secy.

Thirteenth District Society. Dr. Roy Wilson, Seymour, Pres.; Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Secy.

Fifteenth District Society. Dr. Charles Wise, Naples, Pres.; Dr. George Bennett, 402 S. Bolivar, Marshall, Secy.

Clinics

Blackford Memorial Cancer Lectures. Dr. R. G. Gerard, 509 S. Mirick, Denison, Chm.

Dallas Southern Clinical Society. Dr. C. D. Bussey, Dallas, Pres.; Executive Offices, 433 Medical Arts Bldg., Dallas 1.

International Medical Assembly of Southwest Texas, San Antonio, January, 1959. Dr. Lawrence B. Reppert, 817 South Texas Bldg., San Antonio, Secy; S. E. Cockrell, 202 W. French Place, San Antonio, Exec. Secy.

New Orleans Graduate Medical Assembly, New Orleans, March 2-5, 1959. Dr. Eugene H. Countiss, New Orleans, Pres.; Dr. Maurice E. St. Martin, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Oct. 18, 1958. Dr. J. B. Hathorn, Jr., 1500 8th, Wichita Falls, Chm.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 27-29, 1958. Dr. Herman Fagin, Oklahoma City, Pres.; Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Postgraduate Medical Assembly of South Texas, Houston, July 21-23, 1958. Dr. Charles D. Reece, Pres.; Dr. C. Forrest Jorns, Secy., Exec. Office, 412 Jesse Jones Library Bldg., Houston 25.

Board Examinations

Texas State Board of Medical Examiners, Fort Worth, June 23-25. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Secy; Miss Luanna Knox, Assistant Secy.

MEDICOLEGAL NOTES

Voluntary and Emergency Mental Admissions

As numerous questions are arising in relation to commitment under the recently passed Mental Health Code, it may be of interest to the physicians of Texas to consider in a series of articles some of the questions raised and the answers thereto.

Although some of these questions and answers deal only slightly with the physician's part in the commitment of certain individuals to mental hospitals, they do deal with questions with which physicians are often confronted by patients or by those responsible for such patients.

Voluntary Admissions

What are the necessary steps to be taken for a person to be voluntarily admitted to a mental hospital?

The head of a mental hospital may admit, as a voluntary patient, anyone for whom a proper application is filed. Such admission is discretionary on the part of the head of the hospital and should be made only after a determination, based upon a preliminary examination, that the person seeking admission has symptoms of mental illness and will benefit from hospitalization.

What must this application for voluntary admission contain, and with whom is it to be filed?

The application must be in writing and signed by the person seeking admission. If the person is not of legal age, then the application should be signed by the person's parent, legal guardian, or the county judge. It is necessary that the application state that the patient is agreeing to submit to the custody of the mental hospital for diagnosis, observation, care, and treatment for an initial period of not less than 10 days unless the person is discharged prior to that date. The application must state further that the person is agreeing to remain in the hospital until he is discharged or until the expiration of 96 hours after written request for release is filed with the head of the hospital. This application, when completed, should be filed with the head of the mental hospital to which admission is sought.

What effect does a voluntary admission have upon the patient's rights?

It should be mentioned that the head of the mental hospital, upon application for admission by a voluntary patient, is required by the Mental Health Code to inform the patient and any relative or friend who accompanies him to the hospital of his rights and the effect his voluntary admission has upon these rights.

These rights are as follows:

1. The patient has the right to leave the hospital 96 hours after filing a written request for his discharge. This request for discharge must be filed with the head of the mental hospital and signed by either the patient or someone in his behalf whom the patient has consented to act for him.
2. The patient's right of habeas corpus is not affected by his voluntary admission.
3. Possibly most important as concerns the patient is that a voluntary admission does not affect the civil rights or legal capacity of the patient.

Is there any way in which a voluntary patient can be kept in a hospital for continued treatment once he has filed a written request for a release?

Yes, there are two methods. First, the effect of the request for release can be nullified by the patient filing a written withdrawal of such request for release. Second, if prior to the patient's release there is an application for a temporary hospitalization or petition for indefinite commitment filed, then the patient may be detained in accordance with the provisions dealing with temporary hospitalization or indefinite commitment. (These topics will be dealt with in a subsequent article.)

It should be mentioned, however, that so long as a voluntary patient has not filed a request for release, no action may be commenced for either temporary hospitalization or indefinite commitment.

Emergency Admissions (Involuntary)

When and upon whose authority may a person be committed under the emergency admission procedure?

Any health or peace officer who believes, such belief being based upon the conduct of the person or the circumstances under which he is found, that a person is mentally ill and because of such condition is likely to cause injury to himself or others if not immediately restrained may, without a warrant, take the person into protective custody. Such health or peace officer, without taking the individual before a judge, immediately may transport him to a hospital, either general or mental, and make written application for his admission for emergency observation and treatment.

What protection is provided for a person in instances where this emergency admission procedure is used?

The code provides that no person shall be taken into protective custody without an order of the court, unless there is immediate danger of injury either to himself or others. In the event, however, that the emergency admission procedure is used, it is necessary that an order be obtained from the county court within 24 hours of the time the person is taken into protective custody. This order then shall be served on the nearest of kin, if any, or upon a district judge of the county.

For what period of time can a person be detained at the hospital under the emergency admission procedure?

A period of detention is not to exceed 96 hours.

What is done when the health or peace officer delivers the individual to the hospital?

The head of the hospital may admit and detain the person for observation and treatment after obtaining from the health or peace officer who has taken the person into custody a written application stating the circumstances under which he was taken into custody and the officer's belief and reasons for such belief that the person is mentally ill and is likely to cause injury to himself or others if not immediately restrained.

It is also necessary before the hospital may admit and detain the person that there be a written and certified opinion by the medical officer on duty at the hospital, after preliminary examination of the person, that such person has symptoms of mental illness and is likely to cause injury to himself or others if not immediately restrained.

Does the person admitted under the emergency admission procedure have any further protection than that already set forth?

Yes. In addition to the written and certified opinion of the medical officer on duty at the hospital where the admission is made, it is necessary that the head of the hospital have a physician examine every person within 48 hours after his admission to the hospital for emergency observation and treatment and prepare a Certificate of Medical Examination for Mental Illness. A copy of this certificate must then be sent to the person's guardian or responsible relative.

Also, the head of the hospital admitting a person for emergency observation and treatment must give notice immediately of such admission by registered mail to the person's guardian or responsible relative.

—Philip R. Overton, LL.B., Austin.

EDUCATION

Postgraduate Courses

New York University-Bellevue Medical Center's Post-Graduate Medical School, May.—A symposium on dermatology and syphilology, under the direction of Dr. Marion B. Sulzberger, May 19-23; a comprehensive course in cardiology, under Dr. Charles A. Poindexter, May 5-23; an advanced course in urology for specialists, under Dr. Robert S. Hotchkiss, May 12-23; and a symposium on modern therapeutics in internal medicine, under the direction of Professor Charles F. Wilkinson, Jr., June 9-20, will be offered. For further information, write Associate Dean, NYU Post-Graduate Medical School, 550 First Avenue, New York 16.

Geriatric Medicine, St. Louis, May 24-25.—Postgraduate course in geriatric medicine conducted at the Washington University School of Medicine, Division of Gerontology, with special emphasis on heart disease and the psychosocial problems of later life. The course is tuition-free and category I credit will be given members of the American Academy of General Practice. For further information, write to Office of the Dean, Washington University School of Medicine, Euclid Avenue and Kingshighway, St. Louis.

Pediatrics, Philadelphia, May 26-30.—"Pediatric Advances" conducted by the staff of the Children's Hospital of Philadelphia in collaboration with the Department of Pediatrics of the University of Pennsylvania. Curriculum will consist of series of clinics, conferences, and panel discussions on aspects of pediatrics in which important advances are being made. Course has been given category I accreditation by the American Academy of General Practice. Tuition will be $115. Applications should be forwarded to Dr. Irving J. Wolman, Children's Hospital of Philadelphia, 1740 Bainbridge Street, Philadelphia 46.

American College of Physicians, May, June.—Postgraduate courses will be offered on the cardiovascular diseases in the child and adult, May 12-16, University of Illinois College of Medicine, Chicago; internal medicine, June 2-6, State University of Iowa College of Medicine, Iowa City; and hematology for internists, University of Rochester School of Medicine and Dentistry, Rochester, N. Y. Registration fee is $30 for ACP members and $60 for nonmembers. All applications should be made to the American College of Physicians, E. R. Loveland, Executive Secretary, 4200 Pine Street, Philadelphia 4.

Pulmonary Diseases, Saranac Lake, N. Y., June 2-20—Postgraduate course presented by the Trudeau School of Tuberculosis and Other Pulmonary Diseases. Sessions in tuberculosis, silicosis, pulmonary fibrosis, emphysema, fungus infection, sarcoidosis, pneumonias, and intrathoracic tumors will be conducted. All inquiries should be addressed to the Secretary, Trudeau School of Tuberculosis and Other Pulmonary Diseases, Box 500, Saranac Lake, N. Y.

University of Texas Medical Branch

Special visiting lecturers at the University of Texas Medical Branch at Galveston during March included Dr. M. H. Crabb, Texas State Board of Medical Examiners, Fort Worth, "Medical Practice Act of the State of Texas, Medical Licensure, and Problems of Reciprocity"; R. L. Murphee, Ph.D., Oak Ridge Laboratory, Oak Ridge, Tenn., "Influence of Ionizing Irradiation on Reproduction"; Dr. Carl V. Moore, Washington University School of Medicine, St. Louis, Mo., "Recent Developments in the Understanding of Thrombocytopenic Purpura"; and Dr. Jean Brachet, University of Brussels, Belgium, "The Biochemical Role of the Cell Nucleus."

Dr. J. P. Gray with Parke, Davis & Company in Detroit spoke on "Critique on Medical Writings," at a pediatrics seminar, March 28.

Fellowship in Ophthalmology

The Department of Ophthalmology of the New York University-Bellevue Medical Center announces the establishment of the Daniel B. Kirby Fellowship for research in ophthalmology. The initial appointment will be for 1 year at a basic stipend of $7,000 and will be subject to renewal for 2 additional years. Application should be submitted before May 1, 1958, to the Dean, New York University-Bellevue Medical Center, 550 First Avenue, New York 16.

Baylor University College of Medicine

Dr. Neil R. Burch, assistant professor of psychiatry at Baylor University College of Medicine, Houston, has been appointed as a Scholar in Medical Science by the John and Mary R. Markle Foundation. The foundation appoints 25 scholars each year throughout the United States and Canada and allocates $30,000 at the rate of $6,000 a year for 5 years to the school where each scholar will teach and do research.

Dr. Fred M. Taylor, associate professor of pediatrics at Baylor University College of Medicine, Houston, participated in a postgraduate seminar, "The Management of Pediatric Emergencies," sponsored by the Anchorage Medical Society, Anchorage, Alaska, and the Lederle Laboratories February 14.

Dr. Maxwell Finland, associate director of the Thorndike Memorial Laboratory and associate professor of medicine at Harvard Medical School, was in Houston February 10-15 as the Taub Visiting Professor of Medicine at the Baylor University College of Medicine. An international leader in the field of infectious diseases, Dr. Finland delivered the annual Taub lecture on "Antibiotic Resistance." He also spoke to members of the Houston Society of Internal Medicine at their luncheon on February 12.

The budget of the Baylor University College of Medicine has grown from $410,857 in 1947-1948 to $1,395,347 in 1957-1958, representing a 240 per cent increase in the budget from 1948 through 1958.

Research grants have grown from $2,900 in 1946-1947 to $1,292,738 in 1956-1957. Tuition was increased at Baylor this year to $897, the first increase in 4 years despite the increased cost of instruction, which has risen 30 per cent during the same period. Baylor underwrites the education of each physician it graduates to the extent of $12,800. This is $3,200 per student per year that must be raised from private sources. The number of students has increased from 293 in 1947-1948 to 322 in 1957-1958.

Mental Health Campaign

In an effort to help the estimated 17,000,000 Americans who are suffering from a mental or emotional disorder requiring psychiatric treatment, the National Association for Mental Health is sponsoring a campaign beginning with Mental Health Week, April 27-May 3, and continuing through the month of May.

Over $1,230,000,000 is spent each year in tax funds to

care for mental patients, $240,000,000 coming from federal taxes and $990,000,000 from state and local government funds. It is estimated that over 1,500,000 working years of life are lost each year by new patients admitted to mental hospitals. This amounts to $3,000,000,000 in potential net earnings for each group of patients admitted each year. If hospitals were well equipped, were well staffed, and could provide adequate treatment, 7 out of 10 patients could recover partially or totally, the association estimates.

Estimated distribution among new admissions in state hospitals includes 23 per cent with schizophrenia, 25 per cent with senile psychosis and cerebral arteriosclerosis, 6 per cent with involutional psychosis, 5 per cent with manic-depressive psychosis, 13.7 per cent with severe mental conditions brought on by alcoholism, 15 per cent with personality disorders, and 6 per cent with psychoneuroses.

Research Foundation Campaign

The Medical Research Foundation of Texas has launched a statewide campaign to enlist wider interest and support in the development of greater advancement in basic research, announced Dr. G. V. Brindley, Sr., Scott and White Clinic, Temple, new president of the foundation.

Dr. Brindley is replacing Dr. F. J. L. Blasingame, who is now the general manager of the American Medical Association. Franklin Lowney, Galveston, is replacing Dr. Robert C. Connor as director of the foundation.

The foundation has made an institutional grant of $5,000 each to the Baylor University College of Medicine and the University of Texas Dental Branch, both at Houston; Southwestern Medical School of the University of Texas and Baylor University College of Dentistry, both at Dallas; and the University of Texas Medical Branch, Galveston.

MEDICAL MEETINGS

Postgraduate School Conference

Present day problems of cardiac and pulmonary systems were reviewed at the sixth annual Postgraduate Medical and Surgical Conference held in Temple March 3-5.

The conference, sponsored by the education division of the Scott, Sherwood and Brindley Foundation and the staff of the Scott and White Clinic, is a part of the educational activities of the University of Texas Postgraduate School of Medicine. Dr. Melvin A. Casberg, Austin, vice-president of medical affairs at the University of Texas, was speaker at the conference.

Medical Assistants Convention

Guest speakers at the first annual convention of the Texas Medical Assistants Association, which will be held in San Antonio May 2-4, are Dr. C. W. Tennison, president of the Bexar County Medical Society, and Dr. Bernard H. Bloom, both of San Antonio.

The recently organized group is interested in helping medical assistants in all counties in Texas form their own organizations. The purpose of these groups is to help the doctor's assistant improve her standard of service to him and to the patient.

For further information concerning the convention, physicians may write Mrs. Judy Coleman, 408 Medical Arts Building, Dallas 1.

American College of Physicians

Texans participating in the annual session of the American College of Physicians which is being held in Atlantic City, N. J., April 28-May 2 include Dr. Edward A. Cleve, Dr. Robert S. Nelson, Vasant M. Doctor, Ph.D., and Dr. Julian E. Ward.

Dr. Cleve, Baylor University College of Medicine, Houston, will present "Observations in a Large Outbreak of Epidemic Typhus Fever; Vaccination, Serological Studies, Antibiotic Therapy."

A joint presentation on "The Vitamin B_{12} Content of Human Liver as Determined by Bio-Assay of Needle Biopsy Material" will be given by Dr. Nelson and Dr. Doctor, both with the M. D. Anderson Hospital and Tumor Institute, Houston. "Psychophysiological Effects of Weightlessness" will be presented by Dr. Ward, captain in the United States Air Force Medical Corps, Randolph Air Force Base, Randolph Field.

Academy of General Practice

Dr. Holland T. Jackson, Fort Worth, was inaugurated as president of the American Academy of General Practice at its meeting March 24-27 in Dallas. Record attendance at the meeting was 7,431 with a total of 3,634 physicians registered.

Other Texans honored at the meeting were Dr. James D. Murphy, Fort Worth, who was reelected as speaker of the house for the congress of delegates, and Dr. Talbot Green McCormick, Jr., an intern at John Sealy Hospital, Galveston, who was one of the ten winners of the $1,000 Mead Johnson award.

Among the resolutions adopted by the congress of delegates were the continuation of plans for the establishment of a certifying board of general practice, the urging of immediate action towards possible academy representation on the Joint Commission on Accreditation of Hospitals, and policy statements concerning in-hospital training for practicing physicians, including a suggested plan for a sponsorship program.

Medical Technologist Convention

The Texas Society of Medical Technologists will have its annual convention April 25-26 in Houston at the Shamrock Hilton Hotel. Convention activities will include a cocktail party given by the Harris County Medical Society and seminars in microchemistry technique at the Texas Children's Hospital and in paper electrophoreses and paper chromotography at the M. D. Anderson Hospital.

Medical Assembly of Southwest Texas

Officers and directors for 1958 were elected at the annual business meeting of the International Medical Assembly of Southwest Texas February 25 in San Antonio.

Those elected included Drs. A. O. Severance, president-elect; Dr. John C. Parsons, first vice-president; Dr. Jack M. Partain, second vice-president; and Lawrence B. Reppert, secretary-treasurer; all of San Antonio. Dr. L. Bonham Jones of San Antonio became president.

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Sulfamethazine	0.167 gm.	0.5 gram (7.7 grains)
Sulfadiazine	0.167 gm.	
Sulfamerazine	0.167 gm.	
Emulsified vegetable oil	50%	Approx. 500 calories per 100 cc.
Sucrose U. S. P.	12.5%	

Butylated hydroxyanisole 0.01%, sodium benzoate 0.1% and sodium ethylene diamine tetracetate 0.5% as preservatives.

THERAPEUTIC DOSE: Adults initially—two to three tablespoons (30 to 45 cc.) followed by ½ the initial dose every 12 hours. Children initially—one teaspoonful (5 cc.) for each 10 lbs. body weight (up to 80 lbs.) followed by ½ initial dose every 12 hours.

TWICE-DAILY DOSAGE of triple sulfonamides in an oral fat emulsion vehicle resulted in sustained therapeutic blood levels with good clinical responses

INDICATIONS:
Systemic and urinary tract infections due to streptococci, staphylococci, pneumococci, H. influenzae, K. pneumoniae, meningococci, E. coli, B. proteus, B. pyocyaneus, A. aerogenes, B. paracolon and Alcaligenes fecalis.

DOSAGE:

Children:	teaspoonfuls every 12 hours
20 lbs	1
40 lbs	1½
60 lbs	2
80 lbs	3
Adults:	4

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metaphosphate produced markedly higher blood levels than capsules containing either the corresponding base or the hydrochloride alone. In addition, the average levels derived from the tetracycline base or the chlortetracycline base were higher than those produced by the corresponding hydrochloride though lower than those resulting from the mixture containing the base and sodium metaphosphate. In the study with chlortetracycline[6] capsules containing a mixture of the hydrochloride and sodium metaphosphate were also included in the crossover, and the average levels produced by these capsules were the same as with the mixture of chlortetracycline base with sodium metaphosphate.

Although the enhancement of blood levels of tetracycline by phosphate, either complexed to the tetracycline or mixed with the base or the hydrochloride, thus seemed fairly well established, some doubts still remained because certain reliable observers (including many whose results have not been published) failed to confirm the findings with the materials and methods they used. Further confusion seemed to be added by a subsequent report of Welch et al.,[7] who, in repeating a crossover study with capsules of tetracycline phosphate complex and tetracycline hydrochloride with and without sodium metaphosphate, found much hig[illegible]

"...Tetracycline hydrochloride and citric acid, in an encapsulated mixture, produced higher serum concentrations and greater urinary excretions, and hence better absorption of tetracycline, than any other preparation studied..."

[illegible] were published simultaneously with the last mentioned report of Welch et al.[7] These data were based on thoroughly controlled studies both in rats[8] and in man[9] and include additional findings that serve to explain, fairly conclusively, the various discrepancies that have been mentioned.

The experiments in rats[8] were carried out to study the effects of citric acid, dicalcium phosphate, sodium metaphosphate, food, oil and sorbitol on the serum antibacterial activity produced by the administration of tetracycline hydrochloride or tetracycline base. Citric acid administered in equal weight with tetracycline hydrochloride gave the highest concentrations of all the preparations studied. No enhancing effect was obtained from citric acid when given with tetracycline base. Dicalcium phosphate and food resulted in lower, and sodium metaphosphate in higher, serum antibacterial activity than was observed in their absence. Oil and sorbitol did not interfere with tetracycline absorption.

Dicalcium phosphate is widely used as a filler in various capsules, including those of the tetracyclines. The authors cite a large number of other studies that implicate the presence of calcium ions as the cause of the reduced absorption of tetracyclines and show that citric acid can partially neutralize this effect. The depressing effect of food on the serum levels of tetracycline is likewise explained by the goodly amount of minerals contained in commercial laboratory diets, and they postulate that the multivalent cations may be responsible for the poorer absorption of the drug. The authors could not explain the failure of citric acid to enhance serum concentrations when administered with tetracycline base in contrast to its marked effect when given as the hydrochloride. However, they hypothesized that the ability of citric acid to enhance serum levels of tetra[illegible]cline is [illegible] to its ability to form comple[illegible] [illegible]en unavailable for [illegible]he [illegible]th [illegible]ch

[illegible] last mentioned paper of [illegible] et al.[7] indicates that in their study the capsules [illegible]tracycline hydrochloride, chlortetracycline hydrochloride and tetracycline phosphate complex all contained dicalcium phosphate as a filler, whereas the capsules containing citric acid and sodium hexametaphosphate did not contain any dicalcium phosphate. This could clearly explain the discrepancies noted in that study. Likewise, the inconsistencies in other studies may very well have been due to the presence of calcium as fillers in some of the capsules and not in others.

This, however, fails to explain the most recent findings of Welch and Wright,[10] who compared the absorption of three capsules, each containing 250 mg. of oxytetracycline hydrochloride — one without any adjuvant, one with 250 mg. of citric acid and the third with 380 mg. of sodium hexametaphosphate; no other filler was contained in any of these capsules. In triple

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Editorial.
The New England Journal of Medicine.
258:97-99, (January 9) 1958

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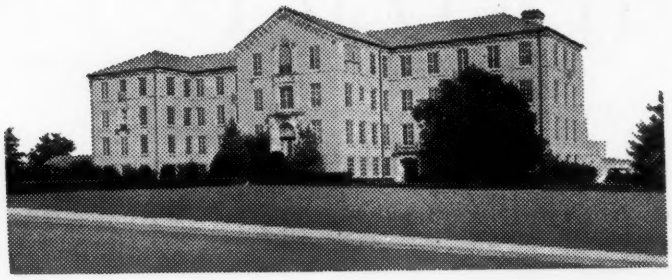

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DRUG NOTES

Recent Urological Solution Dissolves Calcified Calculi

(EDITOR'S NOTE: This is the first of a series of contributions on recent drugs by a member of the University of Texas College of Pharmacy faculty.)

UROLOGICAL SOLUTION G (Abbott) is for the dissolution of calcified urinary calculi. This is Suby's solution G, a sterile solution prepared with citric acid, magnesium oxide, and sodium carbonate; the resulting fluid after interaction contains citric acid along with magnesium and sodium citrates.

This solution is effective in dissolving urinary calculi composed of calcium phosphate, calcium carbonate, or magnesium ammonium phosphate. The primary objective is to keep the solution in contact with the calculus as much as possible. During this time various soluble calcium citrates form. Since the pH is acid (4.0) these are chiefly primary (Ca $[H_2$ Citrate$]_2$) and secondary (CaHCitrate) salts, each of which is only slightly ionized or dissociated into its respective ions. Thus since the primary and secondary salts are soluble and since they are only weakly ionized, calcium ion then is essentially tied up or captured in this soluble form which can be flushed out and the process repeated. The less soluble tertiary calcium citrate ($Ca_3Citrate_2$), some of which is also formed, can be solubilized by the presence of magnesium ion. This is the rationale for the inclusion of the magnesium ion in the formula. A sidelight is that the presence of the magnesium ion also decreased the irritation when compared to solutions containing only citric acid and sodium citrate.

The action of the magnesium may well be one of sequestration. Common sequestering agents are citric acid, citrates, tartrates, glycerin, and even sucrose. These type agents often react with di-valent and tri-valent elements such as iron, copper, magnesium, calcium, and so forth to form soluble, unionized complex compounds. The fact that they are soluble and unionized means that for all practical purposes they are not present in the media for reactions such as precipitation and can be flushed out.

MEGIMIDE (Nicholas), MIKEDIMIDE (Pan Ray) is under clinical trial, not yet available. This substance is beta-ethyl-beta-methylglutarimide, and is proposed as an antidote for barbiturate poisoning.

Mikedimide is listed in the "Veterinary Drug Encyclopedia and Therapeutic Index," both 1956 and 1957, and has been used on animals. The reversal of barbiturate anesthesia is said to be directly proportional to the plane of anesthesia induced almost on a milligram for milligram basis. Poor water solubility of the glutarimide minimizes the danger of overdosage.

Possible uses for this drug may be as a respiratory stimulant in the presence of barbiturates, as routine therapy with barbiturate anesthesia to terminate the anesthesia, to minimize the need for prolonged medical attention while barbiturate anesthesia wears off, or to serve as an effective emergency treatment in the case of barbiturate overdose.

Convulsions have occurred with overdosage, but these can be controlled with barbiturates.

In veterinary practice, the material has been available as a 3 per cent solution in propylene glycol.

Megimide is a very good example of how a small change in chemical structure at times may completely reverse the physiological activity. The barbiturates are malonic acid derivatives. One more carbon atom in this chain gives glutaric acid from which Megimide is derived. Glutaric acid derivatives carrying many of the same side chains present in effective barbiturates have been made; however, thus far only Doriden (Ciba) has become clinically important.

Doriden is alpha-ethyl-alpha-phenyl-glutarimide, the side chains ethyl and phenyl being the same as those in phenobarbital. Theoretically an entire alpha, alpha series, corresponding to known effective barbiturates, could be made and should have sedative-hypnotic activity. When the substituted grouping is moved over one, to the beta position, and at the same time the size of the side chain is reduced, the result is a barbiturate antagonist, beta-methyl-beta-ethyl-glutarimide (Megimide).

PHOBEX (Lloyd, Dabney & Westerfield) is an anticholinergic drug available in tablets of 1 mg. (blue, scored) and 5 mg. (chartreuse, scored). It is indicated in anxiety and tension states, phobias, endogenous depressions, and parkinsonism. Dosage is 1 mg. four times a day initially, with hospitalized psychotic patients or in parkinsonism up to 5 mg. three times a day but not more than 20 mg. daily.

Phobex is benactyzine HCl. It also is available as Suavitil (Merck Sharp & Dohme).

DEPROL (Wallace) is another recent addition to the benactyzine group. Deprol contains benactyzine HCl 1 mg. with meprobamate 400 mg. Meprobamate is Miltown (Wallace) or Equanil (Wyeth). We can think of Deprol as Miltown with Suavitil. Deprol is suggested specifically for depression.

TRIAMCINOLONE is the generic name for Aristocort (Lederle), which has been available since about the first of the year. This fluorine containing steroid has been shown to have a dramatic anti-inflammatory effect and is useful for rheumatoid and inflammatory arthritis.

Significantly there is no sodium retention nor water retention. As with other hormonal steroids there is a slight loss of N, Ca, P, and K.

Kenacort, recently introduced by Squibb, is also triamcinolone and is available in 1 mg. and 4 mg. potencies.

ULTRAN (phenaglycodol) (Lilly) is being investigated as an anticonvulsant.

Previous research has shown that mephenesin [DEP, Tolserol (Squibb), Dioloxol (Carnick)] has anticonvulsant activity but is very short in its duration of action, possibly because of the vulnerability of its two hydroxy groups. Numerous attempts to lengthen this activity, by slight molecular modification, have led to such compounds as Prenderol (Squibb) and eventually to the carbamates Miltown and Equanil.

Recently, Lilly and Company researchers during an investigation for anticonvulsant activity of the closely related ethylene glycols also tested several butanediols. Among these latter were compound 26 [2-(meta-chlorophenyl)-3 methyl-2,3-butanediol] and its para analogue, compound 25 or Ultran (phenoglycodol) (Lilly). Ultran was found effective against both electroshock and Metrazol induced convulsions. This means that Ultran may be effective against both grand mal and petit mal types of epilepsy. Ultran was of longer duration of action but less potent than its para brother. Compound 25 [2-(meta-chlorophenyl)-3 methyl-2,3 butanediol] was the most effective of the entire series of ethylene glycols available. Thus the meta isomer of Ultran (Lilly) may soon be one of our new products.

—HERBERT SCHWARTZ, M.S., Austin.

OF GENERAL INTEREST

Personals

Dr. S. Byrd Longino, Sulphur Springs, was honored by the Hopkins County Memorial Hospital Association on his sixtieth birthday. Friends and relatives presented a "This Is Your Life" program.

Drs. John E. Skogland and *Jack P. Abbott,* both of Houston, were guest speakers at the Rapides Parish Medical Society Founders Day Program in Alexandria, La., January 18.

Dr. Perry C. Talkington, former colonel in the United States Army, has been appointed to the Advisory Editorial Board for the volumes on the "History of Neuropsychiatry, Medical Department, United States Army, in World War II." The 15 member board governs the preparation of the volume.

Recent arrivals include a son born to *Dr. and Mrs. Woolworth Russell,* Amarillo, and a daughter born to *Dr. and Mrs. W. F. Henniger, Jr.,* Galveston.

Dr. Joe C. Rude, Austin, recently visited Air Force Medical Service installations in Germany, France, England, Morocco, and Libya, where he conducted lectures and teaching sessions. He presented "Radiographic Contributions to the Diagnosis of Acute Abdominal Conditions" for the United States Air Force in Europe Medical-Surgical Conference in Wiesbaden, Germany.

Dr. Michael E. DeBakey, chairman of the Department of Surgery at Baylor University College of Medicine, Houston, delivered the eleventh annual Rudolph Matas lecture, sponsored by the Beta Iota Chapter of Nu Sigma Nu fraternity at Tulane University School of Medicine, New Orleans, on March 14.

Potency Plant Investments

Texas Medical Association members approached to invest in corporation stock selling pega palo, a plant reported to improve potency, should make a thorough investigation prior to making any investment, advises Philip R. Overton, general counsel for the Association. The plant has been placed on the prohibited drug list by the Pure Food and Drug Administration, and the Post Office Department has announced the issuance of several fraud orders against several companies transporting the drug.

Mr. Overton calls the attention of Association members to recent promotion in Texas which is attempting to sell physicians $5,000 of original stock and an interest in the franchise for the sale of pega palo, which is being made available on prescription as an experimental drug under the name "Tintura de Pega Palo Fortidom."

A story in the April, 1958, issue of *True* magazine describes the plant, which is native of the Dominican Republic, as resembling poison ivy. *True* refers to pega palo as Rhynchosia pyramidalis. A report on the plant in the *Journal of the American Medical Association* (October 12, 1957), states that the drug has been described to the AMA Bureau of Investigation as Rhynchosia serpentina, then as Paullinia sorbilis or cupana, and later as Rhynchosia pyramidalis. JAMA states that "so far as is known, no active plant principle under any of these designations has been isolated or identified or been demonstrated to have any effect whatever as a drug."

Investigations conducted by *True* show that pega palo is manufactured by the Laboratorio Químico Dominicano in Ciudad Trujillo, under the direction of Dr. José G. Sobá, the Dominican Republic's Secretary of Public Health. Tests, using the drug in 142 impotence cases, according to Dr. Sobá, indicate that 92 per cent of the patients were restored to potency. The *True* report says that 94 physicians in 21 states in this country are conducting investigations with pega palo extract.

Pega palo is being distributed in the United States by a Chicago and a Dallas company. Prepared commercially, pega palo is an alcohol extraction of the dried plant. One ounce of the extract can be mixed with a fifth of liquor. A "shot" is usually prescribed daily.

Further information may be obtained from the United States Pure Food and Drug Administration of the Department of Health, Education and Welfare, 1114 Commerce Street, Dallas; the Pure Food and Drug Division of the State Health Department, Austin; and the Postal Inspector, the United States Post Office Department, 400 North Ervay, Dallas.

Scott and White Clinic Announces Appointments

The Scott and White Clinic and Scott and White Memorial Hospital, Temple, announce the appointment of Drs. Ernest J. Nelson, Abilene, to the Department of Anesthesiology; Walter H. Jarvis, Jr., Grand Saline, Department of Radiology; and Sherman B. Lindsey, Phillipi, W. Va.; Robert C. Little, Norwalk, Ohio; and John R. Shell, Lawrenceville, Va., Department of Internal Medicine.

London Physician Speaks

The second Leslie and Helen Jacobs Foundation Lecturer for 1958 was Dr. John McMichael, professor of medicine and head of the Department of Medicine at the University of London Postgraduate Medical School, who spoke at Southwestern Medical School of the University of Texas, Dallas, on April 2.

The project was sponsored by the Dallas Heart Association and Southwestern Medical School. Funds received were given to the Leslie and Helen Jacobs Foundation. The lecturer is given $1,000 as well as traveling expenses.

Medical Essay Contest

Physician medical writers, who are members of the American Medical Association and residents and citizens of the United States, are eligible to enter the Mississippi Valley Medical Society Annual Essay Contest. Contributions must be on subjects of medical or surgical interest, including medical economics and education. The winning essay receives a cash prize of $100, a gold medal, a certificate, and an invitation to address the annual meeting of the Mississippi Valley Medical Society. Deadline is May 1. Further details may be secured from Dr. Harold Swanberg, 209-224 W. C. U. Building, Quincy, Ill.

Medical Examiners to Give Examinations

The Texas State Board of Medical Examiners will hold its next meeting at the Texas Hotel, Fort Worth, June 23-25 for the purpose of giving examinations and considering applications for licenses by reciprocity. Applications for reciprocity must be complete and on file at least 30 days prior to the June meeting date. Applications for examinations must be on file at least 10 days prior to the meeting.

Medical Research Society Begins New Programs

Three new programs were launched at the annual meeting of the National Society for Medical Research which was held February 11 in Chicago.

A conference program aimed at bringing more consistency and logic to the various state and local laws governing medical research has been planned. A new program launched by the NSMR has the purpose of improving the design of laboratory animal quarters. Another new activity being carried out by the society is a program to educate the public on the importance of student experiments with animals.

The society plans to bring together leaders of many scientific, professional, and cultural groups in a national conference on the legal environment of medical science.

Fellowship Available For Research in Ophthalmology

The Daniel B. Kirby Fellowship for research in ophthalmology has been established by the Department of Ophthalmology of the New York University-Bellevue Medical Center. The initial appointment will be for 1 year at a basic stipend of $7,000 and will be subject to renewal for 2 additional years.

To be eligible for this award, candidates must have had a thorough grounding in ophthalmology, roughly equivalent to a 3 year residency or should have acquired equivalent competence in a basic field related to ophthalmology.

Fellows will be expected to continue work on basic problems in such fields as ocular chemistry, physiology, pathology, or experimental surgery, and will be supplied laboratory space and facilities in the center. Application should be submitted before May 1 to the Dean, New York University-Bellevue Medical Center, 550 First Avenue, New York 16.

Psychiatric Speakers Bureau

Names of psychiatrists who will serve as guest lecturers are being made available by the American Psychiatric Association and the American Academy of General Practice, who are jointly sponsoring the General Practitioner Education Project. One of the services of the project is a speakers bureau, which will give interested medical societies, hospitals, and other medical groups, names of psychiatrists willing to give lectures while taking their vacation trips.

Those interested may write General Practitioner Project, American Psychiatric Association, 1785 Massachusetts Avenue, N. W., Washington, D. C.

Public Health Training Offered

The Public Health Service is accepting applications from professional health workers for graduate training in public health for the 1958-1959 academic year. The program was set up by Congress 2 years ago to help relieve the shortage of trained personnel in public health agencies.

In the President's budget this year, $2,000,000 is requested for the program in the fiscal year 1958-1959 to give additional training to physicians, nurses, sanitary engineers, and other professional personnel whose skills are required in modern public health practice and who are interested in making this field their career.

In addition to tuition and fees, the traineeships provide stipends covering the living expenses of the trainees and their legal dependents. Applications should be submitted to the Division of General Health Services, Public Health Service, Washington 25, D. C.

Review of Polio Events Given by President

Four events were listed in January in a review of poliomyelitis in 1957 by Basil O'Connor, president of the National Foundation for Infantile Paralysis. These included the massive vaccination promotion last spring and summer, the consequent drop in paralytic polio, the expanding research projects of the March of Dimes organization, and the program, "Operation Comeback," sponsored by the National Foundation to bring benefits of modern rehabilitative techniques to many thousands of polio patients.

"Though 1957 was marked by a tremendous campaign, sponsored by the National Foundation and the American Medical Association, to vaccinate the American public against paralytic polio," said Mr. O'Connor, "a shocking number of United States citizens are still unprotected."

The Public Health Service estimated that as of December 1 only three out of five Americans in the susceptible age group under 40 have had one or more injections of Salk vaccine.

During the Mothers' March on Polio which climaxes the January March of Dimes campaign, National Foundation volunteers distributed 30,000,000 polio vaccination reminder and report cards urging parents to give their youngsters, teenagers, and themselves the protection of the Salk vaccine.

Booklets to Help Crippled Children

Booklets designed to help parents of crippled children, but equally applicable to those of children who are not handicapped, have been published recently by the National Society for Crippled Children and Adults.

Earl Miers, editor and Civil War authority, has written his own story of conquest of cerebral palsy in "Why Did This Have to Happen?" Dr. Grace Langdon, a leader in the field of child development and specialist in the relation of toys to everyday living, has written the second, "Your Child's Play." The booklets are available at 25 cents each from the society, 11 South La Salle, Chicago 3.

Twenty-two additional booklets, two more scheduled for publication in 1958, will comprise the Parent Education series. These will be on subjects such as the young child in the family, orthopedic handicapping conditions, problems of adolescence, and emotional growth in parents.

Congresses to Be Held in Mexico

The third Latin American Congress on Obstetrics and Gynecology and the third Mexican Congress on Obstetrics and Gynecology will be held June 8-14 at the School of Medicine of the University of Mexico in Mexico City.

The Latin American Congress is being sponsored by the Latin American Federation of Obstetrical and Gynecological Societies and the Mexican Congress is sponsored by the Obstetrical and Gynecological Mexican Societies. Physicians may register until April 15. Those interested may write Dr. Luis Castelazo Ayala, Secretary General, Ave. Horacio 1506, Mexico 5, D. F., Mexico.

Hospital Administrators To Receive Insurance Bulletins

A series of bulletins entitled, "Health Insurance—Information for Hospitals," published periodically by the Health Insurance Council, will be distributed to nearly 4,000 hospital administrators in general short-term hospitals throughout the country and to executive secretaries of state and local hospital organizations.

The bulletin will analyze the nature and types of health insurance coverage, according to Louis A. Orsini, vice-chairman of the Health Insurance Council. Additional copies of each bulletin may be obtained upon request from the Health Insurance Council, 60 John Street, New York 38.

Award for Best Monograph

The editors of *Modern Medical Monographs,* a quarterly publication, have announced the Medical Monograph Award to be presented for the best unpublished manuscript for a short book on a clinical subject in the field of internal medicine.

The purpose of the award is to stimulate young physicians to communicate their work in the classical form of the monograph and to achieve high standards of medical writing.

The winner will receive $500 and his monograph will be published as a book in the series *Modern Medical Monographs.* Further information may be received by writing Dr. Irving S. Wright, Editor-in-Chief, 450 East 69th Street, New York 21.

Safety Program Planned

The American College of Surgeons, the National Safety Council, and the American Association for the Surgery of Trauma are planning a joint action program aimed at preventing accidents and improving care of accident victims.

Main aspects of the program are public education in accident prevention and handling of the injured, formulation of safety plans for local communities, possible registration of unusual cases of injury, investigations of emergency care of traffic injuries, model legislation to require first aid training for ambulance attendants, policemen, and firemen, and improvement of training and instructional aids.

Law and Medical Institute

A Law-Medicine Research Institute, the first of its kind in the United States, has been established at Boston University to work out a program of research and training in the interdisciplinary areas of law, medicine, and the behavioral sciences. It also will act as a clearinghouse for information on activities in the medicolegal area.

The institute will begin its program by sponsoring a lecture series and by conducting courses in medicolegal subjects.

Pediatric Laboratory Established

A new Pediatric Metabolic Laboratory has been established in the Polio Annex Building of Jefferson Davis Hospital in Houston in memory of William Scott McFarland by his friends and family. It will be used for research into metabolic and renal diseases in children.

Patients and Personnel List Noise in Hospitals Most Annoying

Hospital personnel polled on the nursing care of patients report many times more omissions in care as the patients themselves do, according to a survey of hospital patient and personnel opinion published in the December issue of *Hospitals,* journal of the American Hospital Association.

Among personnel, nursing administrators and supervisors reported the highest number of unfulfilled needs and doctors the smallest number, said the writers of the article. The survey was conducted by the Public Health Service with the cooperation of the American Hospital Association among nearly 9,000 patients and 10,000 hospital administrators, doctors, and nursing personnel in 60 general hospitals.

Both patients and personnel reported noise in hospital rooms and corridors most frequently in their check lists of omissions in care. Both also noted complaints about cold food served to patients, patients awakened too early, and poor ventilation in hospital rooms. It was found that the average nurse spends only 18 minutes with each patient in her unit during the morning shift, and 8 minutes with each patient on the afternoon and evening shift.

Atomic Photographs Help To Diagnose Internal Cancer

Atomic photographs are enabling Veterans Administration doctors to diagnose internal cancer that cannot be found by other methods, the chief of the Veterans Administration atomic medicine program in Washington, D. C., Dr. W. Edward Chamberlain, has announced.

The technique was first applied at the Veterans Administration center in Los Angeles and now is proving increasingly helpful in the care of patients at Veterans Administration hospitals across the nation, said Dr. Chamberlain. The pictures are called scintigrams and are made with a device known as a scanner that records radiation from doses of radioisotopes, said Dr. Chamberlain.

Dots are formed on the paper inserted into the scanner from impulses from radioisotopes within the patient's body. The dotted pictures indicate the areas of concentration of radioisotopes so that doctors may estimate the size of internal organs and diagnose the presence of cancer.

Infantile Paralysis

The National Foundation for Infantile Paralysis has received a grant of $54,090 to support the educational program in the Department of Rehabilitation. The grant is for 1 year and will be used to teach the concept and basic techniques of rehabilitation to graduate and undergraduate medical students.

Physician Members in Civic Clubs

Dallas physicians were recognized in the February 21 issue of the American Medical Association's *Secretary's Letter.* The publication reported that the Dallas Chamber of Commerce has more physician members than any other chamber in the United States.

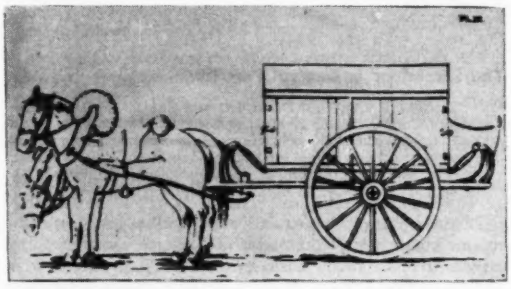
Membership of doctors in the Dallas chamber has grown from 92 in 1953, when only 3 served on chamber committees, to the present with total of 340 physician members, 34 of whom serve actively on committees.

MEMORIAL LIBRARY

Baron Larrey: Military Surgeon

Among the great surgeons of all time stands Dominique Jean (Baron) Larrey. Nowhere in medical annals do we find a more remarkable combination of greatness of achievement and nobility of character than his life presents. His fame and fortunes are inseparably linked with those of Napoleon, whom he served, and as the latter is known as the greatest captain of modern times, so Larrey has been called the greatest military surgeon that ever lived.

Larrey was born in 1766 in southern France. His uncle, Alexis Larrey, a surgeon, gave him his medical education. At 21, he went to Paris and soon was sailing as a naval surgeon to Newfoundland. His career of military surgeon began in 1792 when he joined the Army of the Rhine, and he participated thereafter in 26 campaigns which carried him from one end of Europe to the other and even to Egypt. His relationship to the Emperor of the French has been beautifully portrayed by Dacosta.* "He followed Napoleon with love that never failed, with constancy that never faltered, and gave all his best skill to friend and foe



The "flying ambulance," invented by Larrey, saved many lives during war time. [After Bechet, P. E.: Jean Dominique Larrey; Great Military Surgeon, Ann. M. Hist. 9:429 (May) 1937.]

alike, as duty called. He was brave on the battle field and in the plague hospital, but he was braver in the Tuileries and in the imperial tent; for he always dared to tell his master the truth. That master loved and trusted him; ennobled him; decorated him before the army; when at St. Helena, referred to him in his will as 'the most virtuous man I have ever known,' and bequeathed to him ten thousand francs as a souvenir of enduring affection."

Larrey was a close observer and had a keen insight into the principles of sound surgical treatment which seems remarkable to us today. His memoirs tell of tetanus, of the beneficial effects of maggots in wounds, of brain operations, of goiter and cretinism, of typhoid fever, and of many other phenomena of medical interest. The greatest of his many achievements included the principles he worked out and put in practice in reaching, removing, and caring for the wounded on the battle field. He introduced "flying ambulances," which were two-wheel carts drawn by a pair of horses and contained mattresses, padded sides, and pockets. In warfare, the ambulances saved the lives of countless thousands. At Austerlitz, practically all of the wounded were dressed on the field of battle.

With the fall of Napoleon, Larrey's fortunes declined and he became impoverished. He was restored to the army, however, and died in line of duty in 1842 at the age of 76: "A soldier, a patriot, a great, learned and brilliant surgeon; a brave, truthful and loyal man, a gentleman; and a benefactor to the human race."*

Quotations from Baron Larrey: A Sketch. Papers and speeches of J. Chalmers Dacosta.

Suggested Reading

Bechet, P. E.: *Jean Dominique Larrey; A Great Military Surgeon,* Ann. M. Hist. 9:428-436, 1937.

Brown, M. W.: *Jean Dominique Larrey, Military Surgeon and Humanitarian,* Med. Rec. N. Y. 154:382-384, 1941.

Daly, J.: *Dominique, Jean Larrey,* Med. J. & Rec. 129: 223-282, 1929.

Edgar, J. D.: *Baron Larrey, Medical Officer,* Mil. Surgeon 59:291-307, 1926.

Larrey, D. J. (R. Alexander, translator): *Relation, Historical and Surgical of Expedition of Army of Orient in Egypt and in Syria,* Mil. Surgeon 99:323-337, 790, 1946; 100:68, 175, 256, 342, 428, 523, 1947; 101:66, 144, 232, 321, 516, 1947.

Munroe, A. R.: *Baron Larrey, Surgeon General to Napoleon's Army,* Canad. M. A. J. 48:145-148, 1943.

Orton, H. B.: *Dominique, Jean Larrey, Interesting Facts in Life of Famous Surgeon,* J. M. Soc. New Jersey 48:267-271 (June) 1951.

Phalen, J. M.: *Doctors at Arms, Baron Larrey, Surgeon to Emperor,* Mil. Surgeon 99:337-339 (Oct.) 1946.

Platt, W. B.: *Biography,* Maryland M. J., 31:159-164, 1894.

Wiese, E. R.: Larrey: *Napoleon's Chief Surgeon,* Ann. M. Hist. N. S. 1:435-449, 1929.

—T. H. THOMASON, M.D., Fort Worth.

GIFTS TO THE LIBRARY

Dr. T. J. Archer, Jr., Austin, 75 journals, 38 pamphlets.

Dr. S. W. Bohls, Austin, 52 journals.

Dr. Walter C. Goddard, Austin, 19 books, 70 journals.

Dr. Homer Goehrs, Austin, 4 books, 2 journals.

Dr. Henry L. Hilgartner, Jr., Austin, 3 books, 24 journals, 17 reprints.

John K. Kirby, Ph.D., Austin, and Dr. J. W. Jackson, Kerrville, 7 bound journals, 46 journals.

Bill Lambkin, San Antonio, rare old medical book and kit of surgical instruments.

Dr. A. D. Pattillo, Austin, 24 journals, 6 reprints.

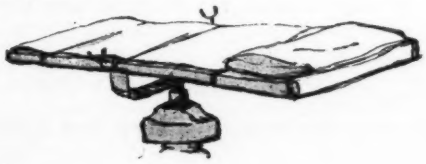
Dr. Morris Polsky, Austin, 5 journals.

Dr. Marion Primomo, Dilley, 76 journals.

Dr. Nelson L. Schiller, Austin, 39 journals, 2 books, 21 reprints.

Dr. B. O. White, Austin, 10 journals.

Dr. David R. Womack, Austin, 70 journals, 30 pamphlets and reprints.



★ Motion Pictures

Borderline

Sound, black and white, 30 minutes, 1955.

"Borderline" is a case study of a 15 year old girl in a remedial center for adolescent girls. Not given sufficient guidance by her parents, she is allowed to date a man who is too old for her. When she returns home late, she is treated with punishment which is withdrawn when she cries. Eventually she is sent to an institution where she and her mother receive professional guidance.

Career: Medical Technologist

Sound, color, 23 minutes, 1954.

The film tells about medical technology through the experience of a young girl and three high school science classmates, all of whom decide to become medical technologists. The key techniques and routines are demonstrated, with explanations as to why they are needed and what they accomplish. Emphasis is on the importance of thorough training.

★ Books

Books Newly Acquired

Alpers, Bernard J.: *Vertigo and Dizziness* (Modern Medical Monographs), New York, Grune and Stratton, 1958.

Ausubel, David P.: *Theory and Problems of Child Development,* New York, Grune and Stratton, 1957.

Conn, Howard F., Ed.: *Current Therapy—1958,* Philadelphia, W. B. Saunders, 1958.

Danowski, T. S.: *Diabetes as a Way of Life,* New York, Coward-McCann, Inc., 1957.

Encyclopaedia Britannica, Britannica Book of the Year, Chicago, Encyclopaedia Britannica, Inc., 1958.

Florey, Howard: *General Pathology,* ed. 5, Philadelphia, W. B. Saunders, 1958.

Glorig, Aram, Jr.: *Noise and Your Ear,* New York, Grune and Stratton, 1958.

Harvey Lectures—1956-1957, New York, Academic Press, 1958.

Morawitz, Paul: *Chemistry of Blood Coagulation,* Springfield, Ill., Charles C Thomas, 1958.

Reich, Walter J., and Nechtow, Mitchell J.: *Practical Gynecology,* ed. 2, Philadelphia, J. B. Lippincott Company, 1957.

Ross Pediatric Research Conference: *Consciousness and the Chemical Environment of the Brain,* Columbus, Ohio, Ross Laboratories, 1958.

Welch, C. Stuart, and Powers, Samuel R., Jr.: *The Essence of Surgery,* Philadelphia, W. B. Saunders, 1958.

Who's Who in America, Vol. 30, Chicago, A. N. Marquis Company, 1958.

Year Book of Drug Therapy, 1957-58, Chicago, Year Book Publishers, 1957.

Year Book of Radiology, Chicago, Year Book Publishers, 1957.

Book Notes

Essentials of Fluid Balance

D. A. K. BLACK, M.D., F.R.C.P., Reader in Medicine, University of Manchester. 127 pages. $3.75. Springfield, Ill., Charles C Thomas, 1957.

This small book is added to the already large array of similar works, as a simplified, up-to-date treatise, directed to "the general medical reader and undergraduate."

An initial consideration of the general properties of body fluid—distribution, electrolyte structure of the phases, and the like—is followed by chapters on water, sodium, potassium, anions and acid-base balance, and finally, a brief approach to treatment.

Several notable points deserve comment: first, lucid expression, typical of the British medical author, aids in following the many complex sequences inherent in an exposition of this subject; second, there is a comprehensive reference list; third, the often contradictory data and conclusions are presented without apparent bias; and, fourth, it is admirably current.

The section on water is particularly good, although the pervasive importance of hypo-osmolality situations is perhaps not stressed enough here, nor in the subsequent chapter on sodium. For the practical minded, there is little concrete in terms of therapy; this may be considered a detraction.

The author deserves commendation for a scholarly, masterfully documented presentation of modern work and thought in this field.

—Philip Fischer, M.D., Dallas.

The Specialties in General Practice

RUSSELL L. CECIL, M.D., Professor of Clinical Medicine, Emeritus, Cornell University Medical College, New York; HOWARD F. CONN, M.D., Fellow, Department of Physiology, Baylor University College of Medicine and the Blue Bird Clinic, Methodist Hospital, Houston, editors. 780 pages. Philadelphia, W. B. Saunders Company, 1957.

Edited by an internist and a general practitioner, this volume comprises all the specialties covered by outstanding experts in each field. Each specialist is a professor in a leading medical school. Most of the contributors have done an excellent job of condensing a broad specialty into a concise, well organized chapter. They stress salient points most pertinent to a general practice or present hard to remember facts in handy table form for quick, easy reference. A few chapters are laboriously long, but the volume in general reviews important broad basic facts garnished by a sprinkling of singular personal observations of the various authors.

Any GP who reads this book will find his efforts rewarded.

—H. C. Day, M.D., Boerne.

A System of Ophthalmic Illustration

PETER HANSELL, M.R.C.S., F.R.P.S., F.B.P.A., Director of the Departments of Photography and Illustration, Institute of Ophthalmology and Westminster Medical School, University of London. 114 pages. $5.75. Springfield, Ill., Charles C Thomas, 1957.

Although written primarily for the professional photographer engaged in the practice of medical photography, this handy exposition will find usefulness in the planning and organization of departments of ophthalmic photography in hospitals and medical schools. The physician interested in photography of the eye will find many valuable suggestions

to aid him in the achievement of professional skill in his clinical photographic records.

The book gives detailed attention to the handling of the patient and anatomical considerations in relation to photographic portrayal. A full discussion of the most useful equipment and materials, as well as the setting up of a studio, adequately covers this phase of illustration. Chapters devoted to working techniques in the studio, ward, operating theater, and consulting room, motion picture photography, and special techniques such as stereoscopic photography and keratography compose the main portion of the monograph. A system of documentation and record filing completes the presentation.

Naturally, the superiority of the accompanying illustrations would make those of us who have attempted this type of illustration envious of this author, who appears to know his subject completely. The book fulfills a definite need in this highly specialized field. It can be well recommended as a guide to perfection for both the professional and the serious amateur photographic illustrator.

—Irwin H. Stolzar, M.D., Jacksonville.

The Neurosurgical Alleviation of Parkinsonism

IRVING S. COOPER, M.D., Ph.D., F.A.C.S., Department of Neurosurgery, St. Barnabas Hospital; Assistant Professor of Neurologic Surgery, New York University, Bellevue Medical Center, New York. 104 pages. $8.50. Springfield, Ill., Charles C Thomas, 1956.

The book is a well written and well illustrated monograph summarizing the present status of the neurosurgical therapy of parkinsonism, with particular reference to the newer operations of occlusion of the anterior choroidal artery and chemopallidectomy.

Dr. Cooper first reviews other surgical approaches to the treatment of parkinsonism, including attacks at various levels of the pyramidal tract, such as cortical excision operations by Bucy and Klemme, pedunculotomy by Walker, and spinal cord pyramidotomy by Putnam. He also summarizes various procedures at the level of the basal ganglions, including Narabayshi's procaine and oil injection into the globus pallidus, Spiegel and Wycis' electrocoagulation lesions employing the stereoencephalotome, Meyer's transventicular approach to the pallidofugal fibers, Browder's capsular operation, Fenelon's transfrontal approach to the ansa lenticularis, and Guiot's subfrontal approach.

Dr. Cooper narrates the story of the now famous operation in October, 1952, on a patient with postencephalitic parkinsonism, in which the left anterior choroidal artery was accidentally torn during an approach to the cerebral peduncle. The vessel was clipped, and the patient showed an abolition of tremor and rigidity in the right extremities and a preservation of motor and sensory function. The surgical technique of anterior choroidal artery occlusion is described in detail together with the postoperative care. Dr. Cooper stresses the importance of selecting cases properly, of restricting surgery to patients under 55, of using preoperative arteriography, of performing the surgery under local anesthesia, of using papaverine intravenously during surgery, and of detailed meticulous postoperative nursing care. The results of more than 50 cases are discussed, including an operative mortality of 10 per cent and 3 hemiplegias. Many case reports are given in detail. This operation was capable of producing good results in approximately 65 per cent of properly selected patients with parkinsonism.

Dr. Cooper then describes in detail the operative procedure of chemopallidectomy, consisting of the placement of a lesion in the mesial globus pallidus first with procaine, and then with absolute alcohol or a combination of ethanol and ethocel. Preoperative pneumoencephalography is done, and the globus pallidus puncture is facilitated by a specially designed light weight pallidectomy guide through a temporal approach. A polyethelene canula is allowed to remain in place and a repeated injection of more alcohol is generally added later. This operation can be done in elderly patients with senile orteriosclerotic parkinsonism as well as in the younger age group, but it is not performed in people with severe mental changes, marked deterioration, or advanced bulbar features. Dr. Cooper gives the results of 70 cases of chemopallidectomy, with a mortality rate of less than 3 per cent and many cases are described in detail. Chemopallidectomy was successful in 85 per cent of the cases at the initial operation; however, there have been recurrences, and lasting effects are observed in only 70 per cent of the patients. In general there is an 80 per cent chance of alleviation of rigidity and a 60 per cent chance of alleviation of tremor by this procedure.

Dr. Cooper's contribution is an enormous one in showing conclusively that neurosurgical therapy can relieve tremor and rigidity in 70 per cent of patients with parkinsonism without producing paralysis. The main criticism of the book is that Dr. Cooper's continued efforts and experiences in the field have already made it out of date, and the book, therefore, is in need of revision and modification by Dr. Cooper.

—Harry Starr, M. D., Beaumont.

Modern Perinatal Care

LESLIE V. DILL, M.D., F.A.C.S., Associate Clinical Professor, Obstetrics and Gynecology, Georgetown University School of Medicine; Washington, D. C. 309 pages. $6.50. New York, Appleton-Century-Crofts, Inc., 1957.

The author of "Modern Perinatal Care" has carefully brought antepartum and postpartum practice up to date. Many of the practices in relation to diet, early ambulation, and travel have been modified since World War II. These transformations have occurred from observations of dietary practices forced on many during the war and by mass population shifts in our own country.

The book is well written and covers the subject well. Inclusion of so many reference numbers in the text is somewhat an annoyance in reading. Most of the complications of pregnancy are discussed, and helpful suggestions for treatment are included.

There are three particularly interesting and helpful chapters. The first is the psychology of pregnancy; the second is on the practice of obstetrics and the law; and third is the discussion of the ethics of the Catholic Church.

This book would be an interesting and informative addition to the practitioner's library.

—Glenn R. Wright, M.D., Cleburne.

Manual of Nutrition

Scientific Adviser's Division (Food) of the Ministry of Agriculture, Fisheries, and Food, Great Britain. 70 pages. $3.50. New York, Philosophical Library, 1957.

This is the fourth edition of a book first published in Great Britain in 1945. The original was the work of Dr. Magnus Pyke under the supervision of Sir Jack Drummond, scientific adviser to the British Food Ministry. It was part of a wartime program of nutrition education. The present edition differs substantially from the original and is the work of the present members of the Scientific Adviser's Division of the Ministry of Agriculture, Fisheries, and Food.

The manual has been divided into four parts; each part is the basis for four lectures. It can be interpreted correctly only by an instructor. It is designed to meet the needs of the caterer or a person already possessed of the practical knowledge of cooking. It is a splendid reference for teachers of domestic science and their students and public health workers.

Child Psychiatry

LEO KANNER, M.D., Professor of Child Psychiatry, Johns Hopkins University; with prefaces by John C. Whitehorn, M.D., Henry Phipps Professor of Psychiatry, Johns Hopkins University; Adolf Meyer, M.D., LL.D., Henry Phipps Professor Emeritus of Psychiatry, Johns Hopkins University; Edwards A. Park, M.D., Professor Emeritus of Pediatrics, Johns Hopkins University, Baltimore. ed. 3. 777 pages. $8.50. Springfield, Ill., Charles C Thomas, 1957.

This is the third edition of a book which is almost unique and seemingly has no competitors as a textbook in this specialty of medical practice. The quality is outstanding, and the information is presented lucidly and concretely in terms which should be understandable to medical student, house officer, pediatrician, or the specialist in child psychiatry. It has much to offer. The book is large, attractively printed, and helpfully indexed, and much is in brief chapters and sections covering specific problems. The mention of two children with hemangiomas in the chapter on physical conditions, and the very different meaning of the condition to each, is an example of clear illustration by cases. There are general approaches to broad areas to provide orientation. One might criticize the book as not being sufficiently dynamic or as lacking in more cohesive pulling together of applied knowledge through theory. Had this been done, however, the book might be less acceptable to those outside the field of psychiatry. There is familiarity with European literature and freedom from dogma or magic formulas.

Particularly good is the material on the relation between pediatrics and child psychiatry. A minority of pediatricians are characterized as "alarmists," "polypragmatists," or "demonstrators." Dr. Kanner stresses that these are a minority. He asserts that children are as interesting as the diseases of children.

There is emphasis on a positive approach to child rearing, and success in child rearing is not viewed as simply the avoidance of stresses for a child. This is in worth-while contrast to some current writings.

As noted in his introduction, any such text cannot be all inclusive. With this in mind, it might be noted that more information on autism, on current child psychoanalytic theory, and infancy would be desirable. References to Escalona and her colleagues in the field of infancy, the work of Grey Walter on learning and the electroencephalogram, Sarason on retardation, and the electroencephalogram and biochemical findings of Heath would be desirable. An expanded section on separation theory and its interaction with the Oedipus complex is desirable. Dr. Kanner tends to prefer the older literature. This literature should not be overlooked, but there is perhaps a little too much on the subject of constitution and typology and on descriptive labels such as those included for the various obsessions.

There is a comprehensive section on pediatric neurology which is useful and helps to keep one reminded of neurological disorders which may appear to resemble those of psychological origin.

—John A. Boston, Jr., M.D., Austin.

Hormonal Regulation of Energy Metabolism

LAURANCE W. KINSELL, M.D. 242 pages. $5.25. Springfield, Ill., Charles C Thomas, 1957.

In recent years the magnitude of the dynamic metabolic activities of the body and the intimate relationship of enzyme activity to the maintenance of health has become apparent. The studies which have given rise to this knowledge are complex and require a well grounded understanding of many disciplines such as chemistry, physics, biology, physiology, and pathology, as well as medicine.

This volume is a recording of the proceedings of the Conference on Hormonal Regulation of Energy Metabolism held in 1956. Participating in this meeting were a superb group of men who are at the forefront of this field; men whose interest transcends any single problem and whose basic knowledge is extensive. Thus, the ideas propounded and the data given are well worth consideration. Although the main emphasis is on carbohydrate metabolism and insulin action, the sections on enzymes, the pituitary, the thyroid, and the adrenal cortex are excellently done.

The book stands as a fine review of the available knowledge as of the time of the meeting. The discussion is involved sufficiently that a casual reader probably would find little to keep him interested after the fine preface by Dr. Joslin. However, anyone especially interested in this field and the amazing possibilities it holds forth or anyone like the reviewer, who has done special work in this area, will find the book stimulating, rewarding, and encouraging.

In short, although not a book for the practitioner or student, it offers a lucid, well organized, and stimulating coverage of one of the most vital and progressive areas in medical research. It should be read carefully by anyone particularly interested in energy metabolism, endocrinology, or diabetes or anyone who wants to know in which direction medical research is going.

—Leonard R. Robbins, M.D., Houston.

From Sterility to Fertility; A Guide to the Causes and Cure of Childlessness

ELLIOT E. PHILIPP, M.A., M.B., B.Chir., F.R.C.S., M.R.C.O.G. 120 pages. $4.75. New York, Philosophical Library, 1957.

Quoting the author of the book, "This new branch of medicine to combat infertility is a thrilling one, for the products are not the mere staving off of ill-health, but the very creation of happiness."

The book will not replace the doctor, but it will save the doctor and the patient time by explaining for the lay public some of the known reasons for infertility and how and why the investigations and treatments are carried out. Diagrams and charts by a foremost artist are helpful.

May's Manual of the Diseases of the Eye; For Students and General Practitioners

Twenty-second edition revised and edited by CHARLES A. PERERA, M.D., Associate Clinical Professor, College of Physicians and Surgeons, Columbia University, New York. 378 illustrations. 518 pages. $6. Baltimore, Williams & Wilkins Company, 1957.

This is essentially the same informative text that became a best seller with its first edition in 1900. The editor, Charles A. Perera, has retained the original qualities which has made it a practical and systematic guide for the student and general practitioner of medicine. Obsolete material has been deleted and discussions of new trends added. The 1955 report of the American Medical Association on computation of visual efficiency loss has been substituted to bring the section on eye injuries up to date.

This is a valuable reference for students and practitioners.

Host-Parasite Relationships in Living Cells

A Symposium sponsored by the James W. McLaughlin Fellowship Program, University of Texas Medical Branch, Galveston, April 27, 1956. Compiled and edited by HARRIET M. FELTON, M.D. 245 pages. $6.50. Springfield, Ill., Charles C Thomas, 1957.

The James W. McLaughlin Fellowship Program is sponsored by a member of the McLaughlin family in honor of his father, who was a member of the faculty of the University of Texas Medical Branch from 1897 to 1905. Dr. McLaughlin was author of "Fermentation, Infection and Immunity," a new theory of these processes, published in 1892.

The subject of host-parasite relationships as seen in living cells seems to be an appropriate theme for this first symposium. The papers are presented by investigators chosen because of their unique contributions in special disciplines and the application of the conventional fundamental sciences to the dynamic three-dimensional environment of the living cell.

This volume is an important addition to the researcher's library.

Digitalis

Compiled and edited by E. GREY DIMOND, M.D., Director, Cardiovascular Laboratories, University of Kansas Medical Center, Kansas City, Kan. 255 pages. Springfield, Ill., Charles C Thomas, 1957.

This book is a compilation of papers presented at a 2 day postgraduate course given by the University of Kansas Medical School. The material is augmented by 3 additional papers from outside contributors.

The entire clinical and investigative field of digitalis is not covered, but most of the important phases are pinpointed.

The latest work on the physiological action, fate, and deposition of digitalis occupies half the book and the rest deals with the clinical use of the drug.

If no other portions are read, the papers by Dr. Batterman on the clinical use of digitalis and Dr. Lown on potassium and digitalis make the purchase worth while. The doctor who gives just a "little bit of digitalis" will better understand the benefits derived from adequate digitalization and the dangers inherent in incorrect usage.

—George E. Clark, Jr., M.D., Austin.

The Gist of Obstetrics

H. B. ATLEE, M.D., F.R.C.S. (Ed. & Can.), F.I.C.S., Head of the Department of Obstetrics and Gynecology, Dalhousie University, Halifax, N. S., Canada. 327 pages. $6. Springfield, Ill., Charles C Thomas, 1957.

The author states in his foreword that this book has grown out of a set of mimeographed notes which were supplied to his medical students. He has given up formal didactic lectures some time ago, but in his foreword does not mention what replacement he has made for the formal lecture.

The book is arranged in the traditional order of most books on obstetrics. It is written primarily for the medical student and as a handy reference work for the general practitioner. It is definitely not a book for the research physician, nor for one preparing for examinations beyond medical school level. There is no attached bibliography to the text.

Throughout the book it is stressed that presentation reflects manner of practice in the author's obsctetrical department, though there are points of variance and disagreement —still they are minor. The author tells why he thinks their procedures are the best for them. When questions arise regarding many of the unsolved problems of the specialty the author presents only one or two theories to explain the particular phenomenon under discussion. This, the reviewer thinks, is a good policy rather than burden one with a review of all the theories that have been devised to account for the particular situation, so many of which theories have been rejected.

Since the book is a compilation of lecture notes, the vocabulary is more informal than one usually encounters, and for this reason the book is easy to read and quite refreshing. The publishers have done an excellent job in their choice of type, and the illustrations are most satisfactory.

—F. W. Lurting, M.D., Big Spring.

Diseases of the External Ear

BEN H. SENTURIA, A.B., M.D., Associate Professor of Clinical Otolaryngology, Washington University School of Medicine, St. Louis, Mo. 211 pages. $8.50. Springfield, Ill., Charles C Thomas, 1957.

This book is a well organized presentation of diseases of the external ear. It includes a detailed classification of all the diseases of the external ear and presents the basic etiologic factors responsible for the bacteriologic, clinical, and pathologic changes observed in external ear diseases. Included is a report of 5 years of coordinated laboratory and clinical research oriented to throw some light on this widely prevalent and aggravating group of diseases.

Thorough consideration is given to the prophylaxis of external otitis with effective proven therapeutic techniques which the author has found successful in treating diseases of the external ear. The book should be of special interest to the dermatologist as well as the otolaryngologist.

—John H. Arrington, M.D., Wichita Falls.

Microtechniques of Clinical Chemistry For the Routine Laboratory

SAMUEL NATELSON, Sc.M., Ph.D., Chairman, Department of Biochemistry, Rockford Memorial Hospital, Rockford, Ill. 484 pages. $11. Springfield, Ill., Charles C Thomas, 1957.

This book is especially valuable to investigators interested in the effect of disease or drugs on the human organism. It is of exceptional value to those faced with the analysis of inorganic and organic constituents present in small amounts in all fields of chemistry.

This book is the outgrowth of brochures which were distributed at demonstrations by the author, the first in 1950 at the convention of the American Chemical Society; the second in 1952 at the American Medical Association convention. The thousands of requests which followed these exhibitions convinced the author of the need for a manual of practical microprocedures for the clinical laboratory.

Terms and their significance are explained in detail and the appendix contains microphysical measurements, normal values, statistical procedures for evaluating test data, absorption spectrophotometry, and chelate compounds.

Roots of Modern Psychiatry; Essays in the History of Psychiatry

MARK D. ALTSCHULE, M.D., with the collaboration of Evelyn Russ. 5.75. 184 pages. New York, Grune and Stratton, Inc., 1957.

Dr. Altschule has written a contribution to medical history which should help modern psychiatrists to orient themselves in a field where disorientation is not exclusively limited to the patient. It is easy to assume that modern concepts were introduced in full blown form. This author examines present concepts of anxiety, ego-psychology, unconscious thought, psychosomatic medicine, and dream interpretation and places them in perspective in regard to their long antecedent history. Acceptance of the writer's viewpoint should help us to be critical of the concepts we use and to be willing to look for something better.

The last portion of the book is controversial. Altschule rejects Freud's concepts and does so on the basis of faulty logic, of faculty scholarship, and of their being little more than Greek philosophic thought presented in a newer dress. A distinction is not made between rejecting Freud's philosophy or scholarship while at the same time accepting his clinical contributions because of their confirmation in clinical practice.

Whether one wishes to challenge the writer on the validity or lack of validity of Freud's concepts, this book is refreshing

in its originality, its scope, and the vast amount of study and thought which it expresses. This meaty survey of the background of modern psychiatry should be particularly helpful to those who struggle to inculcate knowledge of psychiatry in students of medicine.

—John A. Boston, Jr., Austin.

The Medical Interview: A Study of Clinically Significant Interpersonal Reactions

AINSLIE MEARES, M.B.B.S., B. Agr. Sc., D.P.M. 117 pages. $3.50. Springfield, Ill., Charles C Thomas, 1957.

The Human Brain: From Primitive to Modern

A. M. LASSEK, M.D., Ph.D., Professor of Anatomy, Boston University School of Medicine, Boston. 242 pages. $4.75. Springfield, Ill., Charles C Thomas, Publisher, 1957.

Science Looks at Smoking; A New Inquiry Into the Effects of Smoking on Your Health

ERIC NORTHRUP. Introduction by Harry S. N. Greene, Chairman, Department of Pathology, Yale University. 190 pages. $3. New York, Coward-McCann, Inc., 1957.

★ American Medical Association

Rural Health Conference

Representatives of medicine, farm groups, governmental agencies, and other groups discussed rural health problems at the thirteenth annual Conference on Rural Health, sponsored by the American Medical Association Council on Rural Health, held March 6-8 in Jackson, Miss.

Problems in nutrition, dentistry, safety, doctor-patient relationships, physician distribution, health service costs, and the changing patterns of rural society were discussed.

Texas doctors participating in the conference were Dr. W. L. Wilson, Texas State Department of Health, Austin, who participated in a safety panel, and Dr. Dick K. Cason, Hillsboro, who participated in the presentation of "Some Nutritional Target Areas as the World Turns."

AMA Publishes Booklets

A new leaflet, "Look Before You Leap," is being produced by the American Medical Association's Physicians Placement Service as a check list for physicians seeking a location. This publication will be distributed in adequate supplies to state societies, hospitals, and medical schools.

The AMA also has reprinted a series of articles dealing with various health problems of the aging in booklet form from *The Journal of the American Medical Association*. Copies of the series are available upon request from the AMA's Council on Medical Service.

Hawaii Medical Conference To Follow AMA Convention

The Hawaii Medical Association has extended an invitation to doctors to attend the Hawaii Summer Medical Conference in Honolulu, July 1-3. The conference will follow immediately the American Medical Association's annual meeting in San Francisco, June 23-27.

Breakfast panels and a special afternoon clinic at a local hospital will be included in the program. Aside from attendance at the scientific sessions, various other official social functions will be provided in the official trips. A choice may be made of traveling round-trip by air or combining air and steamer travel between the Mainland and Honolulu.

For additional information on conference registration forms or reservations, contact Lee Kirkland Travel, Medical Tours, P. O. Box 3433, Chicago 54.

★ County Societies

Few County Societies Welcome Newcomers to Their Area

Only three county societies have a definite program for welcoming newcomers to their town or area out of the 47 which answered a questionnaire sent to secretaries of county medical societies from the Texas Medical Association headquarters in Austin.

The *Tarrant, Nueces,* and *Harris County Medical Societies* reported having a program for greeting new people to their cities although each society does so in a different way.

Tarrant County Society uses the Fort Worth Welcome Service, which is a private enterprise, as a medium through which to distribute literature to newcomers. The service has proved satisfactory for the 6 years it has been in use. Figures given to the society by the service show that the literature reaches an average of 445 families monthly.

Welcome Wagon leaflets—a short welcome statement telling of medical facilities and a sticker for the phone book—are distributed by the *Nueces County Society* to new people who come to Corpus Christi. *Harris County Society* gives a Newcomers Kit, distributed for the society by the Merchants Greeters Service. The cost for distributing the kits to approximately 400 new families per month is $12.50. The kit contains pamphlets concerning such subjects as the American Medical Association, emergency medical care, medical schools, the family doctor, and the "quack" doctor who guarantees a cure.

Tarrant County Society receives four or five calls weekly from newcomers requesting the name of a family doctor, and *Harris County Society* handles approximately 300 calls each month from individuals requesting referrals to physicians in various fields of practice.

A few societies reported that they offer to help new people find a family doctor. *Dallas County Society* gives complete information concerning a physician's background, training, age, and the like when questioned, and the *Comal County Society* distributes a list of names and addresses of physicians to newcomers. New residents of Corpus Christi are given several names of general practitioners and specialists by the *Nueces County Society*. Upon request, the *Tarrant County Society* gives names of physicians in the vicinity along with their educational background. *Dallas* and *El*

Paso County Societies reported that they make an effort to suggest doctors who will best fit a particular family's needs.

Newcomers learn how and where to get emergency service by various means, such as through the neighbors, local hospital, yellow pages of the telephone book, and doctors. Those societies which have a definite program reported distributing information on emergency service when the newcomer arrives in town.

Some of the societies call to the attention of newcomers the local blood bank and Blue Cross-Blue Shield insurance.

Most of the county societies which give information to newcomers reported that they find it worth while. *Nueces County Society* indicated that several persons have expressed their appreciation to the Chamber of Commerce for distributing the material put out by the society.

County Societies Elect

The following officers were elected at recent county society meetings:

Bosque-Hamilton—Dr. Charles C. Cleveland, Hamilton, president-elect and vice-president; Dr. W. T. Holder, Clifton, secretary and treasurer; Dr. V. D. Goodall, Clifton, delegate. Dr. S. L. Witcher, Clifton, became president.

Brazos-Robertson—Dr. Thomas T. Walton, Bryan, president; Dr. John E. Marsh, Jr., Bryan, vice-president; Dr. Joseph W. Geppert, Bryan, secretary-treasurer; Dr. J. G. Sanders, Bremond, delegate.

Coleman—Dr. Joseph C. Young, president; Dr. Roy F. Kemper, vice-president; Dr. Morris D. Mann, secretary-treasurer; Dr. Roy R. Lovelady, delegate, all of Coleman.

Medina-Uvalde-Maverick-Val Verde-Edwards-Real-Kinney-Terrell-Zavala—Dr. George M. Allison, Uvalde, president; Dr. T. B. Samsel, Jr., Crystal City, vice-president; Dr. George H. Herrmann, Del Rio, secretary-treasurer; Dr. Dean P. Dimmitt, Uvalde, delegate.

Milam—Dr. Travis C. Green, Rockdale, president; Dr. Elmer O. Smith, Jr., Cameron, vice-president; Dr. Philip M. Young, Rockdale, secretary-treasurer.

Montgomery—Dr. Louis F. Brignac, Jr., president-elect and vice-president; Dr. Melvin G. Hutchins, secretary-treasurer and delegate, both of Conroe. Dr. Irving M. Watson, Jr., Conroe, became president.

County Society News Briefs

Dr. Denton Kerr, Houston, president of the Texas Medical Association, spoke on "Are Physicians Meeting the Challenge?" to the *Potter County Society* at its January meeting. For its February meeting, the society heard Donald Anderson, Austin, assistant executive secretary of the Texas Medical Association, who spoke on "Private Practice of Medicine and the Legislative Battle for 1958." The Potter society has made available to lay organizations the film, "Cancer of the Cervix."

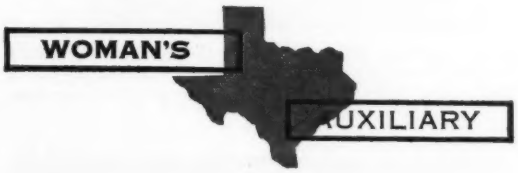
Guest speaker at the March scientific meeting of the *Harris County Society* was Dr. L. K. Diamond, associate professor of pediatrics at Harvard and director of research hematology at the Children's Medical Center in Boston, who spoke on "The Use and Abuse of Transfusions of Blood and Its Products."

J. T. Boothman, chairman of the Texas Pharmaceutical Association's Legislative Committee, spoke to the *Tarrant County Society* on the enforcement and interpretation of the federal narcotic law at its regular meeting February 4. The Tarrant County Pharmaceutical Association has made available to society members a list of narcotics, stating requirements in the filling of prescriptions. The Fort Worth Heart Association presented the scientific program at a recessed meeting of the society February 23.

Guests at the *Tom Green-Eight County Society* March meeting were Buddy Smith, director of the West Texas Pharmaceutical Association, and Eric Anderson, president of the San Angelo Pharmaceutical Society. Requirements in the filling of narcotic prescriptions were discussed. The society will pay trip expenses to the Brownwood Science Fair for 4 children, 2 first place and 2 second place winners from the San Angelo Science Fair.

Dr. Milton Spark presented "Carotid Sinus Syncope, Case Report and Discussion" as the scientific program of the February meeting of the *McLennan County Society*. The society will cooperate with the Texas State Department of Health, City-County Health Unit, Waco Public Schools, McLennan County Schools, and the Tuberculosis Association in the sponsoring of a chest roentgen-ray program in Waco. Dr. Moreton A. Magid spoke on "Disaster—Operation Waco," demonstrating transceiver equipment operating in Waco, at the March meeting of the society. Dr. Spark and Dr. Magid both live in Waco.

Dr. William R. Klingensmith, Amarillo, spoke on "Chest Injuries" at the January meeting of the *Gray-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Society.*



Auxiliary Reports Out of This World

Five-four-three-two-one-zero! It has been count down time for county auxiliary presidents. They have been checking up to learn if their projects "got off the ground" and "orbit" successfully.

Annual reports reveal facts that prove their members have served as a "vanguard" and "explored new space" resulting in improved public relations.

The first Texan who tours the moon may have with him a personal health information card, presented by an auxiliary member. More than 75,000 of these billfold cards have been distributed along with more than 50,000 copies of the Family Health Record at fair booths and to service clubs and preschool and school groups. Also, 10,000 copies

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Officers of the Woman's Auxiliary to the Texas Medical Association for 1957-1958: President, Mrs. Horace S. Renshaw, Fort Worth; President-Elect, Mrs. John D. Gleckler, Denison; First Vice-President, Mrs. John H. Wootters, Houston; Second Vice-President, Mrs. J. C. Terrell, Stephenville; Third Vice-President, Mrs. Richard L. Hudson, Corpus Christi; Fourth Vice-President, Mrs. H. O. Padgett, Marshall; Fifth Vice-President, Mrs. O. N. Mayo, Brownwood; Treasurer, Mrs. William C. Barksdale, Borger; Recording Secretary, Mrs. Harold Lindley, Pecos; Corresponding Secretary, Mrs. J. Franklin Campbell, Fort Worth; Publicity Secretary, Mrs. F. C. Lowry, Austin; Parliamentarian, Mrs. Ramsay H. Moore, Dallas; Executive Secretary, Miss Hazel Casler, Austin.

of "What Everyone Should Know About Doctors" were strategically placed by the members.

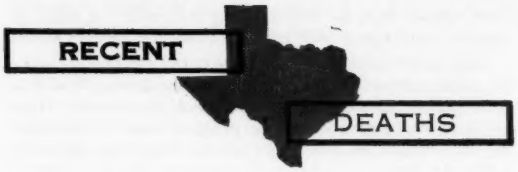
Another sphere of interest continues to be the need for nursing and allied health career recruits. Incomplete reports show 250 girls have entered nursing schools and all are former members of the Future Nurse Clubs, sponsored by the auxiliary members. Gifts and scholarships worth in excess of $10,000 have been underwritten by the auxiliary members to make it possible for more high school students to embark upon health careers.

Science fairs are still in the staging, but early reports listed 6,947 exhibits involving even more students—tomorrow's scientists. Auxiliary members have volunteered to assist in every way, including finances, publicity, transportation, prizes, judging, and as fair hostesses. In areas in which auxiliary members encountered lack of interest, they are persisting and promise results in the future. Those who pioneered this project last year have enjoyed even greater cooperation in their efforts this year and have been recognized for their vision in helping the student scientists.

Bean bags, silver dollars, and fruit cakes are only a few of the missiles that have been projected to make more dollars for the American Medical Education Foundation. The Galveston auxiliary made $659.50 by selling clown bean bag toys. Grayson auxiliary promoted fruit cake sales and netted $400. With a silver dollar as capital, each member of the Harris auxiliary board went to work to make it produce for AMEF. The initial investment was repaid and the ladies went to work making bread, selling violets, baby sitting, and even writing an article for a publication.

These and other achievements to be revealed at the annual convention of the Woman's Auxiliary to the Texas Medical Association in Houston this month will serve as a background against which to launch a new series of projects for the coming year.

County auxiliary presidents' reports at the fortieth annual meeting of the Woman's Auxiliary to the Texas Medical Association in Houston will serve as a challenge to Mrs. John D. Gleckler, Denison, President-Elect (left), and proof of success as President for Mrs. H. S. Renshaw, Fort Worth (right). Mrs. R. C. L. Robertson, Houston (center), was president of the Harris County Auxiliary during the past year in addition to serving the convention as general chairman.



DR. RAYMOND L. BRADLEY, SR.

Dr. Raymond L. Bradley, Sr., of Houston died December 24, 1957.

Dr. Bradley was born July 9, 1892, in Battle Creek, Mich., and was the son of Clarence Leslie and Bertha (Barr) Bradley. He received his preliminary education in a Houston high school and attended Rice Institute and the University of Texas, Austin. He was graduated from the University of Texas Medical Branch, Galveston, in 1918. He interned at St. Mary's Infirmary, Galveston, and served a residency at St. Joseph's Infirmary, Houston. He practiced in Houston from 1919 until his retirement in 1956, giving emphasis to surgery, gynecology, and obstetrics.

He was a member of Harris County Medical Society, Texas Medical Association, and American Medical Association. He also was affiliated with the Masonic Lodge and Shrine. He was in the medical reserve corps of the Coast Guard during World War I.

Dr. Bradley is survived by his wife, the former Miss Edith Beazley, whom he married in 1917 in Houston; a son, Dr. Raymond L. Bradley, Jr.; and two brothers, Rolland Bradley and Earl C. Bradley, both of Houston.

DR. E. J. ARENDT

Dr. Erich J. Arendt, San Antonio, died December 24, 1957, of heart disease.

Dr. Arendt was born in 1879 in Laubegost, Germany. He received his preliminary education in Germany and was graduated from the American Medical College, St. Louis, in 1905. He practiced in San Antonio from 1921 to 1940, when he was appointed to the city health department staff as an epidemiologist specializing in venereal disease control. He retired as a health official in 1957. He was acting health director six times, the last time in 1955.

Dr. Arendt was a member of the Bexar County Medical Society, the Texas Medical Association, the American Medical Association, and the Texas Public Health Association. He had also been a member of the Dallas County Medical Society.

He is survived by his wife and one son.

DR. FRANK F. HEGER

Dr. Frank Ferdinand Heger, San Antonio, died January 19, 1958.

Dr. Heger was born July 12, 1898, in Hallettsville, and was the son of Joseph and Mary (Rabel) Heger. He attended Moulton High School, Texas Agricultural and Mechanical College, College Station, and Southwest Texas State Teachers College, San Marcos, and was graduated from St. Louis University School of Medicine in 1928. He interned at St. Louis City Hospital and practiced in Seguin and Moulton before going to San Antonio in 1938. He served as major in the Army Medical Corps from 1940 to 1945.

He was a member of the Bexar County Medical Society, Texas Medical Association, and American Medical Associa-

tion. He previously had been a member of Lavaca County Medical Society. He also belonged to the Catholic Church, Knights of Columbus, Woodmen of the World, Sons of Hermann, Veterans of Foreign Wars, and the Catholic Physicians Club. He also was an amateur radio operator and photographer.

Dr. Heger and Miss Selma Emery were married on May 3, 1932, in Moulton. Survivors include his wife and two children, Frank Emery and Miss Mary Catherine Heger, both of San Antonio, and ten brothers and sisters.

DR. HOMER B. ALLEN

Dr. Homer Bryan Allen, Brownwood, died January 21, 1958, of coronary artery disease, congestive failure, and diabetes mellitus.

The son of S. B. and Celia E. Allen, he was born February 5, 1891, at May. He attended May schools and Britton's Training School in Cisco. He was graduated from Howard Payne College, Brownwood, in 1914 and from the University of Texas Medical Branch, Galveston, in 1919. He served his internship at Gulf, Colorado, and Santa Fe Hospital in Temple. After practicing a short time at Roby, he moved in 1920 to Brownwood, where he practiced until his death.

He was a past president and secretary of the Brown-Comanche-Mills-San Saba Counties Medical Society; a member of the Texas Medical Association, American Medical Association, Texas Air-Medics Association, and Texas Railway and Traumatic Surgical Association; a fellow of the American College of Surgeons and the Academy of International Medicine; and a certified fellow of the International College of Surgeons.

Dr. Allen was a trustee of Howard Payne College, a past president of the Kiwanis Club, a deacon of the First Baptist Church, and a member of the Masonic Lodge. He served as a private in the Army in World War I and as a lieutenant commander in the Naval Medical Corps Reserve during World War II. He was instrumental in the founding of the Medical Arts Hospital at Brownwood.

He was awarded a certificate of appreciation signed by Franklin D. Roosevelt for "uncompensated service patriotically rendered his country in the administration of the Selective Service System."

Dr. Allen married the former Miss Carrie Elizabeth Camp on September 15, 1918, in Brownwood. She survives as do four children, Samuel B. Allen and Charles J. Allen, both of Brownwood; Dr. H. B. Allen, Jr., McGregor; and Wendell Allen, Long Beach, Calif.; two brothers, E. Otis Allen, Norton, and O. C. Allen, May; and two sisters, Mrs. Perry Wagnon, Temple, and Mrs. V. Earl Earp, Monahans.

DR. I. STANLEY KAHN

Dr. Isidore Stanley Kahn, San Antonio, died January 26, 1958, of complications of diverticulitis.

He was born in Dallas on August 18, 1879, and was the son of E. M. and Lillie (Hurst) Kahn. He received his preliminary education at Sachs Collegiate Institute, New York, and was graduated in 1900 from Harvard, Cambridge, Mass., and in 1904 from Harvard Medical School, Boston. He interned at Boston City Hospital until 1905, at which time he began practice in Dallas. He also practiced in Mexico before going to San Antonio in 1912. He retired in 1953. Dr. Kahn served as major in the tuberculosis section of the Army Medical Corps during World War I.

He was a member of the Texas Medical Association and the American Medical Association through the Bexar County Medical Society. He also was a member of the Southwest Allergy Forum, Southern Medical Association, American Academy of Allergy, and the National Tuberculosis Association and a fellow of the American Academy of Allergy, the American College of Allergy, and American College of

DR. I. STANLEY KAHN

Physicians. He was a thirty-second degree Mason and belonged to the Shrine. He was a director of E. M. Kahn and Company of Dallas. He had written several papers on allergy for medical journals.

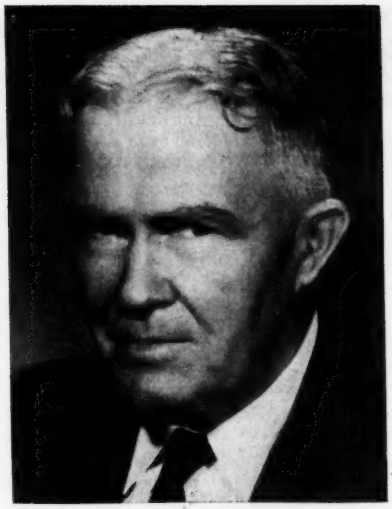
Dr. Kahn and Miss Emma M. Grothaus were married October 30, 1937, in San Antonio. She survives, as do two grandsons and three sisters, Mrs. P. W. Hessberg, New York; Mrs. A. H. Sanger, Dallas; and Mrs. A. F. Weisberg, Sr., Dallas.

DR. ERNEST R. McCAULEY

Dr. Ernest Ruffin McCauley, Moody, died March 5, 1958, of a heart attack.

Dr. McCauley was born August 29, 1881, at Pleasant Plains, Ark., the son of William A. and Ella (White) McCauley. He attended the Stampede school near Moody and was graduated from the Central Texas Institute in Moody in 1902. He attended the Fort Worth University Medical Department, Fort Worth, from 1903 to 1905 and was graduated from Southwestern Medical College, Dallas, in 1908. He did postgraduate work at the University of Texas Medical Branch in Galveston. He practiced at Sadler for 2 years and at Whitson for 14 years before coming to Moody in 1923. He continued his practice in Moody until his death.

He was a past vice-president of the Bell County Medical Society and a member of the District 12 Medical Society, the Texas Medical Association, and the American Medical Association. Dr. McCauley was a local physician for the Santa Fe Railroad, city health officer, and a member of the staff of Hillcrest Hospital, Waco. He was a thirty-second degree Mason and a member of the First Baptist Church,



DR. ERNEST R. McCAULEY

the Lions Club, and the Chamber of Commerce. He had served several terms on the Moody School Board.

Dr. McCauley is survived by his wife, the former Miss Cena Hill, whom he married December 27, 1906. Other survivors include a son, Ray McCauley, McGregor; a daughter, Mrs. Cecil Rutherford, Longview; three sisters, Mrs. A. T. Nelson and Mrs. Nelle Ritchie, both of Denton, and Mrs. Nora Ford, Moody; three grandsons; and two great-grandchildren.

DR. WILLIAM LEE HART

Dr. William Lee Hart of San Antonio, dean emeritus of Southwestern Medical School in Dallas, died December 22, 1957.

Dr. Hart was born January 27, 1881, in Yorkville, S. C., the son of George Washington Seabrook and Ellen Almene (Hackett) Hart. He was graduated from the University of Maryland Medical College, Baltimore, in 1906, and was in residency at Army Medical School in 1907-1908. He did postgraduate work at the Command and General Staff School in 1926, the Army Industrial College in 1927, and the Army War College in 1931. He received a doctor of laws degree from Baylor University in 1945 and a doctor of letters of humanity degree from the Southwestern Medical Foundation in 1946.

He entered the United States Army Medical Corps in 1908 as a first lieutenant and was promoted to a brigadier general in 1945. He commanded the Twelfth Medical Regiment in the Philippine Islands and was president of the Seventh Central American Medical Research Board. He made cholera researches in the Philippines and was honored by the governments of Bolivia, Ecuador, Serbia, Poland, France, Montenegro, Panama, and others. He became dean of Southwestern Medical College in Dallas in 1946, holding that position until 1950, a year after the institution became Southwestern Medical School of the University of Texas.

Dr. Hart was awarded the Founders' Medal by the Association of Military Surgeons of the United States in 1942 and was co-founder of the Celsus Society of San Antonio. He was a member of the American Medical Association, American Association for the Advancement of Science, American Public Health Association, Texas Academy of Science, Society of the Cincinnati, Geographical Society of Mexico, Association of Military Surgeons, Asociacion Mexicana de Medicos Militares, and the Asociacion Nacional de Venereologia.

He was a fellow of the American College of Physicians and the American College of Surgeons, an honorary member of the York County Medical Society in South Carolina, and a past president of the Scientific Society of San Antonio. He had been a member of the Dallas County Medical Society and the Texas Medical Association.

DR. WILLIAM LEE HART

Dr. Hart also belonged to Pi Delta Upsilon; Theta Nu Epsilon; Upsilon Pi; Pi Gamma Mu, social science honorary fraternity; Alpha Omega Alpha, honorary medical fraternity; Nu Sigma Nu, medical fraternity; and Sigma Xi, honorary science fraternity.

He is survived by his wife, the former Miss Mariana Catherine Franklin, whom he married on June 30, 1920; two children, William Lee, Jr., San Francisco, and Mrs. Augustus Clemens, San Angelo; two sisters; and three brothers.

DR. LUTHER F. NAYLOR

Dr. Luther Fines Naylor, Waco, died November 8, 1957, of generalized arteriosclerosis.

Dr. Naylor, the son of James Parker and Sarah Frances (McClain) Naylor, was born August 17, 1877, in Moody. He was educated in the public schools of Moody and attended Baylor University, Waco, and Southwestern University, Georgetown. He was graduated in medicine with honors from the University of Nashville, Nashville, Tenn., in 1905, and did postgraduate work at the New York Medical College, New York, in 1910. He interned in hospitals in Nashville and in the University of Nashville Clinic. He was in general practice in Coryell County, Bell Falls, and Moody before coming to Waco in 1910 as an eye, ear, nose, and throat specialist. He was on the staffs of Providence Hospital and Hillcrest Baptist Memorial Hospital, both in Waco. He retired from medical practice in 1954.

He was a member of the McLennan County Medical Society, the Texas Medical Association, and the American Medical Association. He belonged to the Masonic Lodge, the Knights of Pythias, Woodmen of the World, Praetorians, Waco Chamber of Commerce, and the Austin Avenue Methodist Church. Dr. Naylor played football for Baylor University and was later team physician for an early nineteenth century Waco baseball club.

Dr. Naylor is survived by his wife, the former Miss Harriet Elizabeth Rutherford, whom he married in October 27, 1897; a daughter, Mrs. Arthur E. Curry, Waxahachie; and twin sons, John Rutherford Naylor, Bryan, and James Parker Naylor, Waco.

DR. DAVID O. LOWRY

Dr. David Oscar Lowry, Cooper, died December 16, 1957.

Dr. Lowry was born December 3, 1875, in Pontotoc County, near Pueblo, Miss., the son of Adolphus Carter and Mary Jane (Harris) Lowry. He received his preliminary education at Plymouth, Miss., and attended the Mississippi Normal Institute at Sherman. He was graduated from the Memphis Hospital Medical College, Memphis, Tenn., in 1901. He did postgraduate work at Chicago Policlinic and Hospital in 1916 and in clinics in New Orleans in 1923. Before coming in 1910 to Cooper, where he stayed until his death, he practiced first at Lake Creek and then at Enloe.

He was appointed by the then governor of Mississippi as a hospital steward in the United States Army during the Spanish-American War in 1898 and was with the Medical

DR. DAVID O. LOWRY

Reserve Corps during World War I. He was appointed by Governor Beauford Jester as examiner for the Reserve Selective Service System in Texas in 1948. He also served as examiner for the Selective Service Board in Cooper during World War II.

He was a member of the Lamar County Medical Society, the Texas Medical Association, the American Medical Association, and the American Academy of General Practice. He had been a member and a past president of the Delta County Medical Society, serving eight terms in this office. He belonged to the Methodist Church, the Chamber of Commerce, the Country Club, the Red River Valley Association, and was a past president of the Lions Club. His outside business interests included stock farming and cattle raising.

Dr. Lowry is survived by his wife, the former Miss Lula Roberts, whom he married December 27, 1903; two sons, Weldon C. Lowry, Cooper, and David O. Lowry, Jr., Dallas; and three daughters, Mrs. Mary L. Tompkins, Houston, Mrs. Mildred L. Ewell, Bellaire, and Mrs. Nell L. Smith, Corpus Christi. Other survivors include a brother, James M. Lowry, Pontotoc, Miss., and two sisters, Mrs. W. W. Weatherall, Pontotoc, Miss., and Mrs. Sid J. Harris, Rio Grande City.

DR. KAY B. URBAN

Dr. Kay Bonner Urban, Tyler, died of a heart attack on January 24, 1958.

Born August 30, 1894, in Bullard, Dr. Urban was the son of William B. and Dixie (Oliphant) Urban. He attended Bullard High School; Lon Morris College, Jacksonville; and Southwestern University, Georgetown. He received a bachelor of science degree from the University of Texas, Austin, in 1915 and, after attending the University of Texas Medical Branch in Galveston for 2 years, he was graduated from the University of Pennsylvania School of Medicine, Philadelphia, in 1919. He served his internship at the Germantown Hospital in Philadelphia. He later did postgraduate work in pediatrics at Vanderbilt University in Nashville.

Dr. Urban practiced in Crystal City and Rusk before going to Gonzales, where he was associate medical director of Gonzales Warm Springs Foundation. He later moved to Tyler to become associated with the East Texas Tuberculosis Hospital. He also was at one time director of the American Red Cross Hospital in Seybo, Dominican Republic, and director of Mission Hospital, Shiprock, N. Mex. He was in the Student Army Training Corps during World War I.

He was a member of the American Medical Association, Texas Medical Association, Cherokee County Medical Society, and American Academy of General Practice. He had served as secretary of Medina-Uvalde-Maverick-Val Verde-

DR. KAY B. URBAN

Edwards-Real-Kinney-Terrell-Zavala Counties Medical Society in 1940 and president of Cherokee County Medical Society in 1948. He had been a city health officer. He was a steward in the Methodist Church for 28 years but was a member of the Presbyterian Church at the time of his death. He was a Mason, Rotarian, and a member of the Phi Alpha Sigma medical fraternity and Kappa Alpha social fraternity. His hobby was flower gardening.

Dr. Urban and Miss Carol M. Smith were married June 10, 1919, in Texas City. He is survived by his wife; a son, Robert E. Urban, Port Arthur; a daughter, Mrs. Justin Stewart, Dallas; a brother, Quinn Urban, Tyler; a sister, Mrs. John Gilchrist, Jacksonville; and four grandchildren.

DR. RICHARD C. BERNARD

Dr. Richard Charles Bernard, Dallas, died December 29, 1957, from polycythemia with a leukemoid reaction.

Dr. Bernard, son of Adolfo and Magenta Bernardini, was born November 12, 1903, in Hoboken, N. J. He attended Jersey City grammar schools and private junior and senior high schools in Florence, Italy. He was graduated from the Royal University of Florence, Academy of Medicine, on July 11, 1929. He served his internship at the Bergen Pines County Hospital in Paramus, N. J., and then was assistant superintendent and medical director of this hospital from 1935 through 1939. Specializing in diseases of the lung, he established a private practice in Hackensack, N. J., from 1939 to 1941. He was on the McKnight Texas Tuberculosis Hospital staff in Sanatorium from 1941 to 1947, and returned to private practice at Harlingen from 1947 to 1954, when he retired because of ill health. The following year he became a part-time member of the staff of Woodlawn Tuberculosis Hospital, Dallas, where he remained until his death. He was also a part-time physician for the Veterans' Administration from 1947 to 1954.

He was a member of the American Medical Association and the Texas Medical Association through the Dallas County Medical Society. He belonged to the American Trudeau Society. He also had been a member of the Cameron-Willacy and the Tom Green-Coke-Crockett-Concho-Irion-Sterling-Sutton-Schleicher Counties Medical Societies.

Dr. Bernard was married February 8, 1947, to the former Miss Joanna Shaefer in Oklahoma City. She survives as do his parents, Mr. and Mrs. Adolfo Bernardini, Montecalvoli, Italy, and two brothers, Dr. S. V. Bernardini, Florence, Italy, and James S. Bernard, Jacobs, Natal, Union of South Africa.

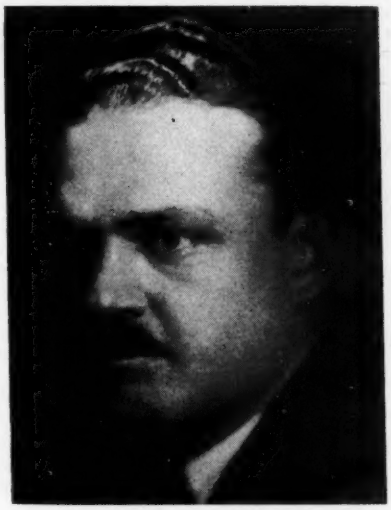
DR. JOSEPH H. McCRACKEN, JR.

Dr. Joseph Hill McCracken, Jr., Dallas, died January 18, 1958.

Dr. McCracken, son of Dr. Joseph Hill and Mary Sue (Wilson) McCracken, was born February 16, 1902, in Mineral Wells. He attended public schools in Mineral Wells and the University of Texas, Austin. He received his degree of medicine in 1926 from the former Baylor University College of Medicine, then in Dallas. He interned and was house surgeon at St. Luke's Hospital, New York, and was a resident at the Hospital for Special Surgery, New York.

He practiced in Mineral Wells for a year before moving to Dallas, where he specialized in general surgery. He had taught special surgery at Baylor University College of Medicine in Dallas before the school was moved to Houston. He was on the staff of Baylor Hospital and the attending staff of Gaston Avenue Hospital.

Dr. McCracken was a member of the Dallas County Medical Society, Texas Medical Association, American Medical Association, Southern Medical Association, Dallas Southern Clinical Society, District 14 County Medical Society, and Dallas Society of General Surgeons, of which he was a charter member. He also was a fellow in the American



DR. JOSEPH H. McCRACKEN, JR.

College of Surgeons. He belonged to the Sigma Alpha Epsilon social fraternity, Phi Chi medical fraternity, Journal Club, Dallas Country Club, and Terpsichorean Club, and was a member of the Highland Park Methodist Church.

He is survived by his wife, the former Miss Mary Frances Hall, whom he married December 22, 1923, in Dallas; a son, Joseph Hill McCracken, III, Dallas; and two sisters, Mrs. Robert A. Ellison, Fort Worth, and Mrs. James Elders, Mineral Wells. Dr. McCracken's father was President of the Texas Medical Association in 1911, and his wife of the Woman's Auxiliary to the Texas Medical Association in 1955.

DR. PAUL M. SIMS, JR.

Dr. Paul Martin Sims, Jr., Beaumont, died December 28, 1957.

Dr. Sims was born July 14, 1924, in Woodville, the son of Paul M. and Pheobe (Crews) Sims. His preliminary education was received in French High School and Lamar College at Beaumont and the University of Texas at Austin. He was graduated from the University of Texas Medical Branch, Galveston, in 1947. He served his internship and residency at John Sealy Hospital, Galveston, from 1947 to 1949, specializing in cardiovascular research. He established a general practice in Beaumont in 1949 and continued this until his death.

Dr. Sims was a member of the Jefferson County Medical Society, the Texas Medical Association, and the American Medical Association. He was a captain in the United States Army in Munich, Germany, in 1954-1955.

He is survived by his father, Paul M. Sims, Sr. of Beaumont.